# THE JOURNAL

OF THE

OKLAHOMA STATE MEDICAL ASSOCIATION

VOLUME XXIX | McALESTER, OKLAHOMA, JUNE, 1936 | Number 6


# The Mortality of Acute Appendicitis*

## *Consideration of Preventive Measures*


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In a paper presented before the Academy of Medicine in November, 1932, the opening statement is as follows: "Acute appendicitis is a curable disease. If the proper remedy is applied before the inflammation spreads beyond the appendix, the cures should amount to one hundred per cent."

That statement was based on the limited experience and observation of the author. Acute appendicitis, however, occurring as it may in those who have disassociated but serious lesions, will still command treatment. These disassociated lesions may, in certain instances, be of such nature as to make surgical procedures hazardous in themselves. It follows, therefore, that while the statement above quoted may still be true in selected cases, there are complications which will eventually bring about a mortality in simple acute appendicitis.

In the January number, 1936, of *Archives of Surgery*, Schullinger publishes a paper in which he gives the results of acute appendicitis and associated lesions, over a period of eighteen years, in the Presbyterian Hospital in New York. Schullinger's analysis of these cases is, to me, the most enlightening expose of the subject that has ever come to my attention. His studies and his statistics have largely furnished the inspiration by which I undertake to present this paper.

Schullinger divides appendicitis into five groups. The first group is acute appendicitis without any associated lesions, and he gives his definition as follows: "An acute inflammatory process confined almost entirely to the appendix in whole or in part." Out of a total of 2,653 cases, 1,175 fall into this group. The number of deaths in the 1,175 cases was seven, or a mortality of .59 per cent. Two of these cases do not, in my opinion, rightfully belong in this group. Briefly, one was a six year old child admitted to the Hospital with a diagnosis of intussusception; the diagnosis was verified at operation. The appendix was included in the intussusception and on examination after reduction, the operator thought it appeared to be acutely inflamed and removed it. The report from the surgical pathological laboratory was "chronic appendicitis." The patient lived only ten hours after operation. Cross indexing probably explains this case being included.

The second case was that of a twenty-seven year old man admitted to the Hospital because of pneumonia and a failing heart from chronic valvular disease. The abdomen was slightly distended, no tenderness or spasm was present. Patient died five hours later. Autopsy revealed obvious pneumonia, edema of the lungs, cardiac hypertrophy, as well as mitral and aortic stenosis. The abdominal cavity was clean and the peritoneum smooth, pale and glistening. The appendix appeared normal in its proximal two-thirds but the distal

*Read before the Surgical Section, Annual Meeting, Oklahoma State Medical Association, Enid, April 8, 1936.

third was slightly distended and the serosa distinctly congested. Section of this portion of the appendix revealed a few drops of pus within the lumen.

Of the five remaining, one, thirteen years of age, was admitted with a diagnosis of tuberculous peritonitis and chronic ileus. A few days later a rather severe pain developed in the right lower quadrant of the abdomen and operation disclosed an acute inflamed appendix and no evidence of tuberculous peritonitis. Three days later the child developed signs of atelectasis of the left lung, together with bronchial pneumonia. On the eleventh day there was an acute ileus; cecostomy was performed. Following this a chronic cough developed with signs of bronchial pneumonia over the lower lobe of the left lung. Because of the persistence of the cough and pulmonary signs, it was felt that it might be tuberculous, but bacilli were not found on repeated examinations. On the fortieth day severe stomatitis set in, which rapidly spread to all parts of the oral cavity. Patient died on the fiftieth day.

Two fatalities were due to pulmonary embolism, one on the fifth and one on the sixth day following operation. One had an acutely inflamed retrocecal appendix; the other was an obese man, fifty-two, who suffered from marked post-operative distention. The operation had lasted eighty minutes because of difficulty in removing the appendix.

In two other cases, Schullinger states death might have been prevented by the use of spinal anesthetic or avertin, or even local anesthesia. The first was a man of thirty who had a cold and a sore throat at the time of admission. He took ethylene poorly and finally had to have ether by the open method. The operation was long and difficult because of the retrocecal position of the appendix. He developed bronchial pneumonia on the fourth day; coughing provoked disruption of the wound on the eighth day, which was immediately repaired. Death followed in twenty-four hours.

The other was a very obese man of fifty-two, in which the operation lasted one and one-half hours because of high retrocecal appendix. Ethylene ether was used. Post-operative course was stormy because of progressive distention and bronchial pneumonia of the lower lobe of the right lung. Jejunostomy was performed on the fourth day, at which time there was no evidence of peritonitis, and the patient died twelve hours later.

The death rate in this first series was practically unchanged from year to year throughout this eighteen-year period and a graph made on the basis of progressive five-year periods shows practically a constant level.

In group two, there were 630 cases with twelve deaths. Group two is acute appendicitis with acute local peritonitis. The mortality in this series was 1.9 per cent. Studied in progressive five-year periods, there is a distinct falling of the mortality rate in this group. In the 1918 to 1922 period, the rate was almost four per cent. It was more than three per cent in the 1920 to 1924 group, but from there on, there is a steady decline to the 1929 to 1933 group, in which the mortality was less than one per cent. This indicates that during this eighteen-year period, there has been a definite progress made in the treatment of appendicitis associated with local acute peritonitis in the Presbyterian Hospital.

The third group is acute appendicitis with peritoneal abscess. In this group there were 571 cases, total number of deaths sixty, per cent rate is 10.5. Taking again the progressive five-year period, we find that the death rate in 1918 to 1922 was fourteen per cent. The period from 1925 to 1929 was the least, having declined from the previous period to less than eight per cent; but from this group forward to the last period, namely from 1929 to 1933, it rapidly rose and in the last period it was twelve per cent; showing that at least during the last several years no progress had been made, but rather a loss had been sustained.

Group four is acute appendicitis with acute diffuse spreading peritonitis. In this group there were 329 cases, fifty-six deaths; a mortality of 17.2 per cent. The progressive five-year analysis of these cases show no improvement in eighteen years.

Group five is acute appendicitis with acute progressive fibrinopurulent perito-

nitis. In this group there were twenty-five cases and twenty-two deaths, a mortality per cent of eighty-eight. There were thirteen cases in the first nine years with eleven deaths; in the second nine years there were twelve cases with eleven deaths, showing the results in the latter years are not so good as in the previous.

Summarizing, I quote again from Schullinger's paper: "The total mortality rate from acute appendicitis at the Presbyterian Hospital from 1916 to 1933, inclusive, is 5.08 per cent, or 135 deaths in 2,653 cases. The total death rate for each of the five groups is as follows: acute appendicitis 0.59 per cent; acute appendicitis with acute local peritonitis 1.9 per cent; acute appendicitis with peritoneal abscess 10.5 per cent; acute appendicitis with acute diffuse peritonitis 17.02 per cent; and acute appendicitis with progressive fibrinopurulent peritonitis eighty-eight per cent."

Acute appendicitis is not a preventable disease. We have seen from the figures quoted that the mortality of acute appendicitis *per se* is less than one per cent: we have seen that in acute appendicitis, even with local peritonitis, it is less than two per cent: we have seen that the other associated lesions, namely, peritoneal abscess, spreading peritonitis, and fibrinopurulent peritonitis, furnish the major mortality. We have stated that acute appendicitis is not preventable. We can state with just as much certainty that the associated lesions of acute appendicitis which furnish the vast majority of the deaths, are preventable. It remains for us, therefore, to apply the principles of prevention in such manner as to not permit appendicitis to go beyond the first, or certainly not beyond the second stage, as described in the quotations herein given.

It seems as if we have fallen into the wrong way of thinking about appendicitis. The international classification of the cause of death is not according to the usual conception, certainly, of the laity. We are in the habit of thinking that when a patient with appendicitis dies, it matters not what the complication was, as having had a single entity. For example, comparing appendicitis to other diseases and conditions: If a person has repeated attacks of tonsilitis and subsequently develops myocarditis, or septicemia, we think of these as distinct entities, and scarcely associate them with the initial cause. This is strikingly true in tetanus, where, for instance, the initial lesion has been a puncture wound.

If we could learn to think of appendicitis in the same light, namely, that of itself it is not so serious except for its associated lesions and complications, and get this thought over to the laity, we might expect some real progress in our efforts to reduce the mortality. After all, we will have to re-educate ourselves and educate the public, if we are to get the desired results.

As shown by the figures, we are not making progress in those grave cases which we too frequently see in neglected appendicitis, and this, in spite of the fact that for the last many years the major portion of the discusions carried on in medical societies and in the journals have been on the treatment and management of the complications of appendicitis. This is as it should be as long as we are to have these complications, and no one is optimistic enough to think that we will not continue having them. It seems, however, that we should go back again to the simpler question of appendix itself. Since we will admit that the complications of an inflamed appendix can be prevented if the proper treatment is applied in due time, the method by which this may be accomplished is the problem before us.

In this paper we will not discuss the treatment of the complications of appendicitis and will assume that everyone will agree that the proper treatment of an acutely inflamed appendix with, or without, local extension of the inflammation, is operation. We assume that this is now generally accepted as proper, in spite of the fact that we are constantly seeing cases of appendicitis that recover from repeated attacks without operation. The time will never come when any of us will be wise enough to be able to state in what cases this happy result will follow. So we hold that the proper treatment is the removal of the appendix at the earliest possible moment, as long as the disease is limited to the appendix or to its immediate vicinity.

The main question is, therefore, how can we get these cases before the grave com-

plications set in. Obviously it will be through education. First, this education should be among ourselves. The symptom syndrome of acute appendicitis as stated by Murphy many years ago, was about as follows: first, abdominal pain. Note that the location of this pain is not stated other than in the abdomen. To those of us from the south at least, it is best described as belly-ache. This symptom is always present in acute appendicitis. Second, is nausea, which is usually present. Third, toxic signs, usually a temperature elevation; and the fourth symptom in this category is described as localized tenderness in the region of the appendix. It is in this last symptom that we find cause for many errors and delays.

The appendix itself and the visceral peritoneum is not supplied with a sensory apparatus that reacts to inflammation in the form of pain. It is only when the parietal peritoneum becomes involved with the products of inflammation, or inflammation itself, that there is pain or tenderness. If an appendix is so situated that it is not in contact with the parietal peritoneum, the disease may go on to rupture and destruction without there being any localized pain whatsoever.

Where is the surgeon of considerable experience who has not seen cases that presented no localized tenderness before the signs of general peritonitis appeared? The point to be made is, that localized tenderness and rigidity mean at least that the products of the inflammation have gone beyond the appendix, and that if we wait for the sign of local tenderness before we make a diagnosis, in many cases, we will do so at grave risk to the patient. Therefore, it seems that any person who is seized with abdominal pain, who is nauseated, and who presents the signs of toxemia, should be considered as most likely having acute appendicitis, and should at least be constantly observed until the diagnosis is found to be otherwise. If we educate ourselves along this line, and are, therefore, competent to pass this along to those who look to us for guidance and protection of their health, we will begin to make progress.

How shall the public be educated? There is ample proof that it can be successfully done. For five years or more, the City of Philadelphia has conducted an intense campaign along this line and it is stated that the results are striking and that there has been a decided reduction in the mortality of appendicitis in this period. They have educated the druggist not to prescribe over the counter for persons who have abdominal pain, at least, before warning them of the possibilities as to the cause of the pain; they have exhibited placards in various public places, continually reminding the public of the dangers of appendicitis and of its initial signs and symptoms. Lectures have been given to students in the schools and colleges.

Another incident of education, which is closer home, is among the employes of a large corporation, who have been given instruction in health and first aid. The opportunity was furnished in these courses to inform this group of the early signs of acute appendicitis. As a result, we have information direct, that numbers so instructed have gone to physicians when they suffered abdominal pain, for the purpose of having it determined at once, if possible, whether or not they had a diseased appendix as a cause of pain, and, in many instances, it has been found to be the cause.

Education may be conducted by every physician who is in active practice, it matters not what his specialty may be. In making physical examinations, for whatever cause, the physician can remark, without unduly alarming the individual, that appendicitis is a disease which many times strikes without warning; that it may attack anyone, it matters not what their mode of living has been; and that at least the first sign is pain or distress in the abdomen and that this initial pain is not localized in the region of the appendix. We recognize that there is such a thing as appendix phobia. Constipated and nervous individuals are prone to have pain in the abdomen, usually in the right lower quadrant. If, in giving instructions, one will emphasize the fact that the initial pain of acute appendicitis is not in the right lower abdomen, this group of people may be spared considerable worrying.

Then education may be conducted along another line. We will always have a group of people who shudder at the thought of any treatment that implies surgical opera-

tion, and regretfully we must admit that we still have doctors who will try to treat a patient with acute appendicitis in a manner as to avoid an operation. I would like to quote again from Schullinger: "Certain fears and superstitions still exist in the minds of many, that to enter a hospital and undergo an operation is an acknowledgment of defeat, and can result only in certain death. The problem is often exasperating and the practitioner can do nothing but observe the chances of recovery gradually diminishing with the advance of time. When consent to operation is finally given, it may be too late. The practitioner knows only too well that a certain number of cases of acute appendicitis will subside with morphin, rest and the use of the ice bag. He institutes this therapy for twenty-four hours (the most important factor in reducing the mortality is, therefore, lost) and returns the next day. If the patient is better, the therapy is continued and recovery probably takes place. The physician is considered a miracle man; he saved his patient from an operation and "froze" out the appendix. But what if the patient is worse, or only about the same? The physician will decide to send him to the hospital, or else will determine to watch him for another twenty-four hours. In either case, the damage has been done. The patient no longer has a localized process but probably spreading peritonitis due to perforation or gangrene of the appendix. The physician has done "his best;" he has tried to save his patient from an operation and in so doing the patient's life has been jeopardized. So the patient goes to the hospital and either has a long stormy course which may be of great economic loss, or else dies. The result is that the hospital and the surgeon are blamed and the practitioner is completely exonerated. From the standpoint of economy alone, the public should learn that the conservative and economic treatment of acute appendicitis is early, prompt operation."

Recapitulating and summarizing, we can say that acute appendicitis is a disease that shows no favors; it strikes the high and the low; the rich and the poor; the old and young; the weak and strong; none are immune to it.

Appendicitis is a non-preventable disease which carries a mortality of less than one per cent, so long as the disease is confined to the appendix and so long as the appendix is removed before extension takes place. The mortality of acute appendicitis and its associated lesions is, in well organized institutions, about six per cent, and there are about 24,000 deaths in this country annually from these causes. With a mortality rate of acute appendicitis uncomplicated at one-half of one per cent, the total mortality should not be over 2,000 annually. This means that since the associated lesions of appendicitis are preventable, that if the disease is treated while it is still limited to the appendix, there will be a saving of about 22,000 lives annually. Does not this possibility justify our using our best efforts to attain that figure? There is no reason to believe that the incidence of appendicitis will ever be lessened. We need not give any thought to that consideration, but if we put our shoulder to the wheel, we may greatly reduce the mortality that comes from those lesions that are preventable.

We should look upon appendicitis as a disease that demands consideration, it matters not what other co-existing diseases may exist, so long as those co-existing conditions are not hopeless. For example, we might be excused from operating on a person who has acute appendicitis in the presence of the last stages of a malignant disease, or of tuberculosis, but there are many conditions and diseases which do not constitute contraindications. For example, the contagious diseases, infectious diseases, pregnancy, tuberculosis in all but the last stages, etc., are not contraindications. I have gone into a home quarantined because of scarlet fever and operated on a child, in the height of the eruption of this fever, for acute appendicitis. Convalescence was uneventful.

We must change our way of thinking about appendicitis. First, we must look upon simple appendicitis as a disease which usually has very few but constant symptoms. Marked tenderness of the abdomen, particularly with rigidity in any portion, is a sign that complications are about to take place if they have not already done so. Therefore, we must take such steps as we can and as soon as we can, to prevent further extension. We

must learn to look upon appendicitis as a condition which brings about complications which are the real causes of death. Therefore, as a prophylactic measure, we must remove the appendix when it is diseased, in order to prevent those complications.

We should use every opportunity to educate the public in the early signs of this trouble. Appendicitis is not necessarily a public health question, in that it is not a communicable disease, but it is an economic problem and therefore the public should be interested, and this would justify the public health agencies assisting in the campaign of education. One of our recent commissioners of health conducted such a campaign during his incumbency in office.

This is a problem that belongs to all of us, all physicians should be interested and at every possible opportunity when in contact with people education should be carried on.

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## DISCUSSION

*Dr. C. E. Northcutt, Ponca City*: Unfortunately, when we read a paper or hear a discussion on appendicitis we are prone to think of it lightly, but I am glad to see so many doctors here are interested in this most important subject.

Many of us are drawing much surgical work from appendicitis. To many of us, our philosophy of medicine is the happiness we derive from the practice. The more successful we are, the greater the pleasure. The care of patients with pathological appendices forms a large percentage of our practice, so it will be seen that the more successful we are with the care of an appendix, the greater the pleasure that will be derived. It is a well known fact that for every loss we have in this type of work it is necessary to complete a hundred successful cases.

In this paper that has just been read Dr. Reed has very ably classified the types of appendices. We have the acute appendix, the acute appendix with local peritonitis, the acute appendix with appendiceal abscess, the acute appendix with spreading peritonitis, and the mucopurulent fecalith type of appendix.

Now from the percentage point of view, which is the way we must consider our work—bankers consider theirs that way, and we, as physicians, must consider our success on a percentage basis—in the first two classifications the death rate is approximately one per cent. Undoubtedly this is due to early diagnosis and treatment. It should be our aim to always keep the mortality in this type of condition at one per cent or lower. In the other three classifications we have made some mistake, because the majority of complications are preventable. Mistakes are usually a misdiagnosis, or we procrastinate and try to save the patient financially at the expense of his health. When we do this, the case falls into one of the last three classifications where the mortality runs between thirty-five and forty per cent.

Dr. Reed has covered this field very carefully, and in detail, but there are two or three points which I wish to mention.

We think of an appendix as we think of a man's face. All of us have noses, but there are no two noses in this room exactly alike and no facial characteristics exactly the same. The same is true with the appendix. However, I like to classify an appendix in one of two main groups: First, where the base is funnel shaped. The other, where the base is quite narrow.

A patient with the funnel shaped appendix is the type that will go on and have repeated attacks of pain in the side, or chronic appendicitis. The other type is the one with the narrow lumen; an attack in this type of appendix is usually fulminating in character.

Every abdominal pain should be considered as possible appendicitis until proven otherwise. In cases where the appendix is suspected, it is our duty to first convince the patient, and I believe that most of the medical profession will agree, that the only treatment is surgical. The appendix will have to be removed sooner or later; the sooner the better.

Of course, morphine should never be given to a patient with abdominal pain until the diagnosis is made and appendicitis ruled out. If morphine is given, the symptoms will be masked, and where an appendix would otherwise be taken care of in one of the first two classifications it

will fall into one of the latter classifications, with an increase in mortality. A patient with abdominal pain, much less a suspected appendix, should never be given a physic; if there is any doubt, an enema should be given. No one will do a laporotomy in an acute abdomen without complete laboratory examination, including differential count and sedimentation test.

An acute appendix is out on the end of the cecum like an egg on a whip. The gallbladder is more fixed, and when old Mother Nature sees trouble she will throw the omentum around it, but with the appendix loose in the abdomen this is very often impossible, and it cannot become walled off so easily.

I wish to express my appreciation for having the opportunity to hear and discuss this very important paper which was so ably presented by Dr. Reed.

*Dr. Hirschfield*: I would like to make a few remarks on the psychology of this subject. The professional side has been covered very well by Dr. Reed and Dr. Northcutt. I think we should bear reference more to the psychology with the patient and the family. The professional side is pretty well taken care of for we can take the patient to the hospital and the treatment is pretty well standardized. If we get them early there is not much chance but the poorest of us will have success. What this probably needs is more salesmanship. Ordinarily I think perhaps many of us are better salesmen than surgeons, but this is a case where salesmanship is important. If we get them early the operation is simple and safe; I would rather have the youngest interne operate in the first twelve hours than the best of surgeons take the case after complications have developed. There are lots of things we have to combat, and one is the question of irregularity in the patient's diet; that always comes up. Most any of us can go back in our minds twenty-four or forty-eight hours and think of something a little unusual we have eaten. The layman does that during an attack of belly-ache; he thinks of cucumbers, hot dogs or hamburgers or something he has eaten and sells himself the idea that is the cause of his stomach-ache. I don't think I can recall a case of acute appendicitis where the patient couldn't think of something he had eaten in the last twenty-four hours that might not be the cause. I have seen many with a diagnosis of ptomaine poisoning already made by the patient or his family. All young people and most old ones have a diganosis already made. Another thing we should do is to educate the profession that while many of these cases are very typical, I think we have been oversold on the textbook characteristics of appendicitis. Most of us are prone to think that in appendicitis the diagnosis stands out like the nose on a man's face and can't be mistaken by anybody, but there often comprises some syndrome of symptoms that we don't always have. We all want to understand that when we have the typical textbook case we have an advanced case. Temperature, leucocytosis, nausea, tenderness, especially with vomiting, is always an advanced case. If we get these cases early enough to reduce our mortality we should get them before they are so typical that even the internes or students can make a diagnosis. The physician can conscientiously feel that he had better err on the right side. If he is not sure he ought at least to get the patient to a hospital where he can be watched and where they can get laboratory work and with the standpoint of leucocytosis rule out any possible right renal lesion. Our profession has always been very backward about selling itself to the public. We have let the various quacks sell themselves to the public while we have stood back on our dignity and ethics and have not advanced our own ideas to the public. We should educate ourselves to sell ourselves in the practice of the profession. I think in this connection the Medical Society and the American College of Surgeons and even the local societies ought to get out a little leaflet—department stores get to where they definitely check every piece of mail that goes out and their monthly statements, and if they don't weigh enough to make up the limit allowed for postage they add some leaflets—and I think we as a profession ought to do the same thing. We should have little sales talks on the danger of appendicitis authorized and sponsored by the parent societies, the A. M. A. and A. C. S., and inclose this literature in our monthly statements. They would be read and in my opinion would

be an aid; in other words, what we need is salesmanship as well as plenty of good surgery.

*Dr. Dickson:* I am glad Dr. Reed brought out the one point about these patients in what he called an old-fashioned belly-ache. We read in these texbook pictures of pain of acute appendicitis being situated in the pit of the stomach and radiating around the umbilicus and around the lower stomach. That is not correct in all cases. I have found with the first attack of appendicitis that is so, but if you have a patient who has repeated attacks of appendicitis or recurrent appendicitis in which the appendix is thickened and infiltrated, then you may have the pain any place in the abdomen. Then you can have pain in the lower part of the abdomen; you can have pain starting in the right side over the appendix. About the temperature, I don't believe you can go much on temperature, because I was thrown off by that just here the other day. The patient had a temperature of 103 and 103½. This pain was not localized; there was pain in the lower abdomen as low down as it could possibly be. We waited eight hours and operated the case and it was a very acute appendix. It was thick; the walls were thickened. It had that bluish tinge, acutely inflamed, and on the end of the appendix there was a club with a thin wall just keeping from rupturing. The blood count was high. Fortunately we got this case before it ruptured. Another thing about the acute appendix is the morphine. I saw a girl eight years old whom I couldn't tell anything about. She had been sick for three days. We gave her a small dose of morphine, sent her into the hospital, and in four hours we were able to localize the condition. So I believe if you are where you can watch your patient carefully and see them often that a small dose of morphine will help you localize your symptoms. I don't believe, like Dr. Northcutt said, that we should give large doses that mask the symptoms. But I have never seen morphine given in sufficient quantities to relieve the muscle spasm that you would have in an acute appendix. It doesn't take a very large dose of morphine to quiet down the pain in the abdomen with acute appendicitis. Another thing is nausea. This nausea may not come on early. You see a good many people with acute appendicitis, definitely acute, and the nausea does not come on for twenty-four or forty-eight hours later, and generally in these cases when that happens we find one of these thickened appendices, and I believe the nausea means that we have definitely an appendix and when we get that it means that operation should be done.

*Dr. Mark:* There is one question I should like to ask. What does Dr. Reed consider the value of the blood count?

*Dr. Campbell:* I would like to ask this question before I forget about it—what is the opinion of the doctor on chronic appendicitis. Of course, I realize we have chronic and recurrent attacks of acute appendicitis. I am always in doubt about that. Talking about prophylaxis, in the very few years I have practiced medicine I have observed that I have taken out a number of appendices in children whose mothers I had delivered and were still in bed during their maternity days, and it might lead one to wonder whether diet might lead to appendicitis in these children. I would like to just briefly talk about a case I had. I practice in the country and probably run across a few things the men in town don't see. This boy had been sick three months. I have often wondered about the time in a case where the appendix had ruptured and we had acute peritonitis. This boy's father told me he thought the boy had a ruptured appendix because he himself had been operated upon and had had the same symptoms, nausea and vomiting and diarrhea, and this had gone on week after week for three months. He was in a moribund condition. He had bloody diarrhea—he had not failed a day to have a stool—partially dilated pupils, in all a pitiful case. It was the kind of a case that makes you feel crazy and get mad all over. But the interesting part of the case was that when the boy died, through a little pressure on the father I was able to get an autopsy, and found a sanguinopurulent peritonitis with the appendicial abscess you would expect to find, and there were three kinks or adhesions that must have caused obstruction and it was a wonder they hadn't already caused an obstruction. On the right side was a perinephritic abscess; endocarditis, marked on left side,

mitral valve. The spleen was very red and inflamed and there was nephritis in the kidney on the left side and quite a bit of cellulitis. This was to me an eye-opener and answered the question I had often thought about: How long will they live?

*Dr. Reed:* I will talk briefly. This question about the blood count; it is of value in those very few cases in which we are in doubt. We do them routinely but as soon as the blood is obtained the patient goes on to the operating room and we do not wait for the report. In those few cases where it is difficult to say whether it is an inflammatory condition in the abdomen or intestinal upset from other causes, then the blood count may be of value. As to chronic appendicitis, we have abbreviated symptoms of the acute. There is pain in the abdomen without reference to any particular location. A lot of people who have pain in the side get appendicitis phobia. I don't recognize that at all as a sign of appendicitis, and if that is their only sign I immediately tell them they probably are constipated. Chronic appendicitis has just abbreviated, very mild symptoms of acute appendicitis, namely, general disturbance in the abdomen without any pain referred to the locality. I would like to go ahead and discuss it further but time will not permit. I wish to express my appreciation to you gentlemen for the discussion.

# TRAUMATIC SHOCK*

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A Symposium on Traumatic Surgery would not be complete without some discussion of shock. The term shock is not a new one, but is one which has been used very loosely. It has always been mysterious and still is generally misunderstood. It is probable that the expression was originally used to designate a sudden collapse due to a severe wound. Hippocrates did not use the word shock, but he did appreciate the seriousness of wounds. John Hunter discussed shock in 1793. James Lotta in 1795, employed the term shock with the general meaning now applied to it. In 1870, Samuel D. Gross characterized shock as "a rude unhinging of the machinery of life;" and his description of the clinical picture has never been improved upon even though many advances have been made in our knowledge concerning its mechanism.

Through the investigations of many workers, much of the mystery of shock has been clarified, especially since the World War. Our knowledge is still far from complete but we have learned certain facts regarding the cause of the syndrome, its mechanism, and behavior. We have a better understanding of the problem. We anticipate its onset, think in terms of prevention, and attempt to carry out effectual treatment.

Cannon describes traumatic shock "as a general bodily state which occurs after severe injury and which is characterized by a persistent reduced arterial pressure, by a rapid thready pulse, by a pallid or grayish or slightly cyanotic appearance of the skin which is cold and moist with sweat, by thirst, by superficial rapid respirations and commonly by vomiting and restlessness, by a lessened sensibility and often by a somewhat dulled mental state."

E. M. Cowell, during the World War classified traumatic shock into two types: primary and secondary. Perhaps a clearer classification would be immediate and delayed. Primary shock is defined as sudden collapse occurring immediately after severe injury. Primary or immediate traumatic shock is infrequent in occurrence, and in the absence of severe hemorrhage, has been considered due to some derangement of the nervous system. Since Goltz

*Read before the Surgical Section, Annual Meeting, Oklahoma State Medical Association, Enid, April 7, 1936.

produced primary shock in 1863 by traumatizing the viscera of a frog, little knowledge has been added to this problem in which the symptoms of shock are immediate in onset. The cardinal reason for the apathy, pallor, weak thready pulse, cold extremities, low blood pressure and vomiting of immediate shock, has never been interpreted. Primary shock *does* occur but is *rare*. On the western front in 1916, Santy observed the severely wounded during transportation. He found the temperature to be normal, the pulse to be rapid, but of satisfactory tension, and the general state was not disturbing. As Santy expressed it, "The wounded was not yet shocked." Likewise in civilian life we frequently see a similar picture. A severely traumatized patient is admitted. The case may be an extensive burn, multiple fractures, or a case of visceral trauma. The patient is noticeably calm and seemingly collected. He complains very little and is almost indifferent to his injuries. His temperature is normal and though the pulse rate is increased, it is not very rapid. The blood pressure may be within normal limits. To repeat Santy's expression, "He is not yet shocked." Here, unless one is on the alert, mild and unrecognized primary shock may be allowed to progress into secondary or delayed shock.

Secondary shock, however, admits investigation and considerable information has been obtained concerning the factors involved in producing it. The symptoms characteristic of secondary shock have been produced in animals repeatedly, and by several methods: by bleeding, by traumatizing the intestines, by traumatizing large masses of skeletal muscle or by extensive burns. The work of Cannon, Blalock, Underhill, Phemister, Mann, and others has convincingly attributed the symptoms of shock to loss of blood volume as the primary essential phenomenon. This does not mean hemorrhage to the outside where early death usually occurs if the bleeding is not controlled. On the other hand, following trauma to the tissues, there is an unexplained increased permeability of the smaller blood vessels which results in a loss of plasma into the tissue spaces of the traumatized part. The blood pressure is thereby progressively lowered on account of the increasing loss of blood volume. Concentration of the erythrocytes is resultant until there may be a ninety per cent plasma loss and the haemoglobin may be forty per cent above normal. Unless there is a reaction to this plasma loss which means decreasing blood volume and continuous lower blood pressure, a point is reached when the blood supply to the tissues becomes inadequate and the body mechanism fails. The symptoms characteristic of delayed shock appear and death ensues. Analyzed more critically the apathy, pallor, cold skin, sweating, vomiting and weak pulse of established shock are dependent on an inadequate supply of blood to the tissues. The temperature is low. The body metabolism is below normal. When an individual is damp or cold he may shiver or shake which is a heat producing phenomenon. However, when shock is established, the body is unable to react, and the temperature is further reduced by the profuse sweating which is characteristic. The reduction in blood volume is reacted upon by vasoconstriction which maintains the arterial pressure at or near normal. Notwithstanding, there is a decreased cardiac output and if the circulating blood volume continues to diminish the blood pressure declines in spite of vasoconstriction. Finally there is a failure of the vasomotor mechanism and vaso dilatation results. Associated with this process the decreasing blood volume results in concentration of the erythrocytes, viscosity of the blood, capillary congestion, and anemia of the periphery. Anemia means lack of food, lack of water, and lack of oxygen and in the presence of anoxemia there is acidosis. A series of vicious circles presents itself. The increased permeability of the capillaries decreases the blood volume which lowers the blood pressure. The plasma loss creates increased concentration of the erythrocytes and congestion of the capillaries which results in anoxemia and necrosis. Although the vasomotor center is the last of the bulbar centers to fail from anemia, with no improvement in the cerebral anemia and anoxemia, collapse of the vasomotor center occurs.

This is the picture of established shock. It is not the picture we should look for but the picture we should attempt to prevent or abort. When shock has become

established, treatment is of no avail and there is but a momentary pause in the act of death. When the low blood pressure persists, resulting in anemia of the preferential processes and anoxemia, the vasomotor center loses its capacity to maintain vascular tone and no known agent will bring back the blood flow to normal.

We see and recognize primary shock rarely but we know that mild and unrecognized immediate shock may progress into delayed or secondary shock, and though it is hardly possible to entirely prevent primary shock, anything which decreases the primary shock will be prophylaxis against delayed shock.

Therefore, as prophylactic measures to be instituted immediately, the following are of major importance: Remembering that patients who are suffering from recent trauma or who are in a state of impending shock are hypersensitive to every additional trauma such as rough handling, exposure to cold, wet, or any operative procedure; avoid unnecessary handling. Do not place the injured in bed, shortly move him to x-ray, and then rush him to surgery as is too frequently done. Maintain normal temperature by wrapping the patient in blankets, and applying hot water bags between the blankets. Hot liquids by mouth are beneficial if tolerated. Expose the body as little as possible by uncovering one part at a time. Apply temporary splints at once, which lessens the pain, the trauma, the hemorrhage, and impending shock. If there is external hemorrhage a tourniquet should be applied and not forgotten after being applied but removed in less than an hour and re-applied if necessary. Give morphine in large doses and often, as it is the only drug of real value. It relieves the pain and restlessness. It lowers the blood pressure but in so doing lowers metabolism when there is oxygen need for maintenance of chemical changes. Adrenalin, pituitrin, camphorated oil, etc., are of only temporary value. They do not improve the blood volume, but merely increase the arterial pressure temporarily, which is not needed and in reality decreases the cardiac output. Vasoconstrictor drugs decrease the amount of blood to the organs rather than increase it. Fluids should be given by mouth, *per* rectum, by hypodermoclysis, and intravenously. The introduction of some fluid into the blood vessels which will remain there is the ideal sought. That fluid is not known. Fluid tolerated by mouth and rectum is of real benefit. Fluid given intravenously has only a temporary effect in profound shock on account of the permeable capillary walls. Hypertonic gum acacia solution is recommended and given intravenously to maintain blood volume but its real value is questionable. Whole blood transfusion, given early is a very valuable method of replacing blood volume, raising the arterial pressure and at the same time furnishing oxygen to the tissues. Next in comparative value come the saline and glucose solutions which should be given by all routes in large amounts.

When patient is admitted in potential shock, keen judgment is necessary in deciding if and when to operate. Prompt necessary surgery before shock develops may be wise, with transfusion preceding or during the operation. However, if some signs and symptoms of secondary shock are present, the patient is cold, depressed, and even though the blood pressure is not low and the pulse is fair; delay, heat, fluids, and rest will improve the condition before any surgery is done. When prompt surgery is mandatory expose the patient as little as possible. Use extreme gentleness and meticulous haemostasis. Before any surgery is begun remember that surgical interference may augment the shock, cause a sharp fall in blood pressure, vasomotor collapse, and death. Consider the surgical procedure, the hemorrhage, and do not overlook the depressant effect of the anaesthetic.

In conclusion, the term shock used for years and rather loosely is now used to designate a state of exhaustion which has been rapidly developed by psychic, traumatic, toxic, or thermal stimuli. Its primary cause is not known. The clinical picture is a result of decreased blood volume.

The treatment of established shock is neither satisfactory or successful. We should, therefore, attempt to recognize and render intensive prophylactic treatment to all traumatized patients in whom impending shock is suspected. Having an

understanding of the cause, the course, and the effects of traumatic shock, close observation, frequent blood pressure readings, and intelligent and adequate prophylactic measures should be promptly instituted.

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# Gonorrheal Ophthalmia Treatment*

W. Albert Cook, M.D.
TULSA

The treatment of infantile and adult gonorheal ophthalmias has been so unsatisfactory that a great variety of remedies have been tried, which at the beginning, were thought to be a panacea, but one by one they have been discarded and new ones tried. The object of this brief paper is to report four cases treated last year with one of the newer remedies in the hopes that the discussion will bring out something interesting and the experience of others with this and other treatments which well be beneficial to all of us.

Gonorrheal ophthalmia in infants makes its appearance within a week after birth, but the ophthalmia neonatorum which develops after the first week is more usually caused by the pneumococcus or Koch-Weeks bacillus.

A few years ago the protein treatment was adopted almost universally with marked success, but it has now lost, more or less, its standing, although it still has many strong advocates, but my experience has not been satisfactory. The protein injection intermuscularly seemed to work miracles in some cases, but its action is not uniform in all individuals and can be likened to cold serum which seems to be a panacea at times, and again have no more effect than a hypodermic of sterile water. The one risk of milk injections seems to be anaphylaxis, so that it is always wise to have adrenalin at hand and to give 0.6 cubic centimeter (ten minims) hypodermically at the first indication of distress. Gluteal abscesses may occur, but they respond rapidly to the orthodox treatment.

Nevertheless there are several groups of patients in which milk injections are definitely contra-indicated. These are specified by El-Bakly thus: (1) weak and debilitated patients especially mirasmic children; (2) tuberculous patients; (3) patients with kidney diseases; (4) and women in the last months of pregnancy.

One of the first things, when we see a case of gonorrheal ophthalmia, is to protect the other eye. This has been done for years with Buller's eye shield, but in case you cannot secure any of these shields, a very satisfactory one can be made from an old regular x-ray film from which the chemical has been removed. Some prefer that to the Buller's shield as it is not so cumbersome.

Silver nitrate has been used from time immemorial in these cases in all strengths. The original Crede method in the new born was the instillation of two per cent solution of silver nitrate in the eyes at birth as a prophylactic, but it was found that one per cent solution was just as efficient and there was no possibility of an unsatisfactory result.

This method of treatment of the new born has become so popualr that laws have been passed in forty-four states requiring the application of one per cent silver nitrate to the eyes of all babies at birth.

The use of silver nitrate drops in the eyes other than the Crede method is dangerous and may result in more or less infection of the cornea, in case of an abrasion of the epithelium, and no doubt has been the cause of many opacities which we see in larger children, and are told by the mother that same has been there since infancy, and it should never be used ex-

*Read before the Eye, Ear, Nose and Throat Section, Oklahoma State Medical Association, Annual Meeting, Enid, April 8, 1936.

cept by a competent nurse under the physician's instructions, but is often used to brush the lids in various forms of conjunctivitis, but should be followed with a boric or saline irrigation.

The cause of twenty per cent of the blindness was formerly caused by gonorrheal infection but after the adoption of the Crede method, the percentage has fallen very materially and the percentage at this time is very small.

The pannus caused by trachoma appears to establish an immunity to gonorrheal infections and you seldom see a gonorrheal infection in a trachoma patient.

Ice compresses have a very good influence in controlling swelling of the lids and can do no harm even though it is not curative, it is more or less soothing to the inflamed and swollen lids.

The four cases that I previously referred to were in children from nine to twelve years of age, which came to the Morningside Clinic for treatment for ordinary sore eyes. These cases were all girls, but vaginal smears failed to show any gonorrheal infections. But in three of the cases we were able to get a history of gonorrheal infection in some member of the family or household and the children, no doubt, acquired it from using the same linen, or handling objects, door knobs, and so forth, that had become infected from the hands of the adults. One of these cases had both eyes infected upon admission to the hospital, but the other three cases, one eye only was infected and by placing the shield over the good eye, confined the infection to the original eye.

The treatment followed in these cases after the sound eye had been covered with the shield was boric acid irrigations; cold compresses, which were used where there was much edema, and the instillation of 1-3000 solution of merthiolate every two or three hours. The eyes were irrigated with boric solution each time before the merthiolate was applied and some of you may be of the opinion that the boric acid should have credit for the results, but it is doubtful if boric acid alone ever cured a case of gonorrheal ophthalmia.

There did not seem to be any irritation following this treatment, and in one case the nurse misunderstood the order and used 1-1000 solution, in which case the after effects could not be distinguished from the ones that had the weaker solution.

The education of the soldiers during the World War and the health magazines have done a great deal toward obliterating many causes of blindness and I hope that blindness from gonorrheal infections eventually will be entirely eliminated.

* * *

## DISCUSSION

*Dr. Albert N. Lemoine, Kansas City, Mo.*

Recent researches have quite definitely demonstrated that most of our immunity is established through the skin. The past year gonorrheal filtrate which is given intradermally was introduced. Being given intradermally it comes under the category of immungens producing immunity through the skin. In the past year we have used it in several cases of gonorrheal conjunctivitis. One case in an adult having a corneal ulcer in the lower quadrant and another case in an infant which had a perforated ulcer in the same location. In both these cases the corneal condition was immediately arrested and cleared up with negative smears in an unusually short time. The adult had practically no scar left while the infant had only a scar the size of the perforation. Uncomplicated cases were also treated with the gonorrheal filtrate and they cleared up with negative smears unusually rapidly. The gonorrheal filtrate should be given intradermally, beginning with a small dose, in an infant about one-half minim and in the adult two minims, increasing by an equal amount every two days if the reaction is not too severe. The subsequent injection should be increased less or not at all if the local and general reaction is severe.

I strongly recommend its use in complicated cases.

### A Thirty-four Year Record

The Physicians Casualty Association of Omaha has recently published its report for "thirty-four years of uninterrupted usefulness" in which they show payments to physicians for sick and accident claims of $535,052.98—over a half million dollars. The report further shows they have a surplus fund of $1,350,000 as evidence of their financial ability to pay what they promise to sick or disabled members.

# Cyclopropane in Comparison With Other Anesthetic Agents*

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All of you well know that the anaesthetist of this age has a large number of agents from which to select his anaesthesia. We have all heard arguments pro and con for the various agents. Thus, we thought it would be well to make a comparison of a new and coming gas with actual and unbiased statistics in our own cases. We have given to date something over six hundred cases of cyclopropane, and for the sake of comparison, we selected three hundred cases and compared the post-operative morbidity and mortality with a like number of cases over very nearly the same period of time. This series comprises both clinical, private and charity cases. This comparison will be with the most common agents, such as ether, nitrous oxide alone or in combination with ether, spinals using procaine, and ethylene.

In the ethylene series you will note that we are using one hundred and twenty-seven cases instead of three hundred. As we use our ethylene mainly in O. B. cases and did not think it would be a fair criterion to compare this kind of anaesthesia with surgical anaesthesia. We well realize that there is likely to be some controversy over some of our results that will appear in some of the slides. This comparison is as nearly accurate as is humanly possible to make it and it is not intended to boost one agent and condemn another.

For you who are not familiar with cyclopropane, we will give you a brief regime as to its characteristics, etc. It was first administered to human beings in 1929 by Dr. Ralph Waters of the University of Wisconsin. It is a colorless gas with a characteristic odor that to our sense of smell is similar to the odor of chloroform. It is about one and one-half times heavier than air. It is inflammable but no more so than most other agents that are so commonly used, and according to most authorities, it is less explosive in the mixtures used than the other agents. Since it is a gas it is administered by the closed method.

*Read before the Surgical Section, Annual Meeting, Oklahoma State Medical Association, Enid, April 8, 1936.

The number of operations that were done are listed according to systems under the various agents and are shown in the following table:

| | Cyclopropane | Ethylene | Nitrous Oxide | Spinal | Ether |
|---|---|---|---|---|---|
| Upper abdomen | 25 | 4 | 22 | 1 | 1 |
| Lower abdomen | 84 | 75 | 109 | 212 | 67 |
| Head and neck | 23 | | 29 | | 66 |
| Chest | 55 | | 9 | | 18 |
| Perineum-Rectum | 49 | | 26 | 77 | 6 |
| Extremities | 78 | 40 | 81 | 10 | 104 |
| Miscellaneous | 36 | 10 | 24 | | 30 |
| Total | 300 | 127 | 300 | 300 | 300 |
| Supplemented with ether or other agents | 60 | 65 | 244 | 52 | |

In reference to preliminary medication, that is, the narcotic, etc., we do not use as large a dose of the opiate and use preferably scopolamine instead of atropine with cyclopropane. Since the cyclopropane is not a respiratory stimulant we must bear this in mind when writing the preliminary medication, as with the larger doses we are likely to get some respiratory embarrassment.

The carbon dioxide absorption technique was used in the administration of all the gaseous agents in this series, as that has been our usual routine for some time.

The signs of anaesthesia under cyclopropane may be compared to those of ether, differing slightly in a few respects. In contrast to the other anaesthetic agents, the respiration under cyclopropane more

nearly simulates the normal. In the stage of deep or fourth plane anaesthesia the respiration becomes gradually more depressed, and if pushed still further, stops altogether but does not as a rule become irregular or "jerky" as it does with ether. If respiratory cessation occurs, pure oxygen under slight pressure exerted on the breathing bag is sufficient to cause the patient to resume breathing and the anaesthesia may be lightened.

By reason of the high oxygen content in cyclopropane anaesthesia, we feel it has a decided advantage in this respect over the other agents. Even with ether by the open or closed method, we personally feel that the patient does not get all the available oxygen that he gets with cyclopropane. We use approximately eighty per cent or more oxygen with cyclopropane.

In rapidity of induction, cyclopropane is in the same category with the other gases. It lacks the smothering features of nitrous oxide. It lacks that very unpleasant and obnoxious odor of ethylene. Its stage of induction is free from the choking and long unpleasant induction of ether. From those patients who have had the other agents administered to them, as well as cyclopropane, they show their very emphatic preference for the latter.

In reference to the recovery, the patients as a rule are a little slower than are the patients in whom the other gases have been administered to alone, and not in combination with another agent.

The question about surgical relaxation arises. Spinal naturally stands at the top of the list. Unfortunately not all patients can take spinals and not all of them will permit one to be given, and it has its drawbacks as well as any other agent. Ether in most persons' hands stands next, although we feel we are able in a majority of cases to get relaxation of the same degree with cyclopropane, as has been proven in its use in rectal cases and gall bladder surgery. Very exceptionally has it been necessary to add another agent such as ether, chloroform, etc. Nitrous oxide with oxygen offers practically no relaxation for abdominal surgery. Ethylene, as a rule, will "get by" if the surgeon is tolerant of some rigidity. In the way of gases as to potency, relaxation, etc., cyclopropane certainly heads the field. By reason of the quiet rhythmical breathing, we feel that it overshadows ether as far as the ease under which the surgeon operates. In this series of cases it was possible to obtain sufficient relaxation for the satisfactory performance of twenty-five upper abdominal operations, using cyclopropane, twenty-two under nitrous oxide and ether, nine under open ether and one under spinal. This latter in reference to the spinal can perhaps be explained in that spinal anaesthetics during this series were mainly confined to operations below the umbilicus. In our series in the lower abdominal cases, two hundred and twelve, or thirty-eight per cent, of the cases were done under spinal, eighty-four, or sixteen per cent, under cyclopropane, one hundred and nine, or twenty per cent, under nitrous oxide and ether, and the remainder equally divided under open ether and ethylene alone or in combination with ether.

The pulse rate is slower and more nearly normal when using cyclopropane as compared to other inhalation agents and somewhat resembles that observed under spinal.

In making a comparison of the post-operative complications occurring following the use of various anaesthetics, we realize that many factors are to be considered, such as the type of operation, the amount of hemorrhage occurring, the amount of trauma to the tissues, the duration of the operation, and many other things.

We feel, however, that since the percentage of pre-operative complications was very nearly the same in each group, we can determine to some extent what part the anaesthetics played in the final results.

The major post-operative complications are listed in the following tables:

| | Cyclopropane 300 | | Ethylene 127 | | Nitrous Oxide 300 | | Spinal 300 | | Ether 300 | |
|---|---|---|---|---|---|---|---|---|---|---|
| No. Cases | Maj. 220 | Min. 80 | Maj. 100 | Min. 27 | Maj. 179 | Min. 124 | Maj. 220 | Min. 80 | Maj. 200 | Min. 100 |

## POST-OPERATIVE COMPLICATIONS

### RESPIRATORY SYSTEM

| | | | | | |
|---|---|---|---|---|---|
| | Spinal | Nitrous Oxide | Cyclopropane | Ether | Ethylene |
| Pneumonia | 1.66% | 1.33% | .66% | .66% | None |
| | Ethylene | Cyclopropane | Nitrous Oxide | Spinal | Ether |
| Bronchitis Pharyngitis | 15% | 7.6% | 6.6% | 6.6% | 5% |

### GASTRO-INTESTINAL

| | | | | | |
|---|---|---|---|---|---|
| | Ether | Ethylene | Cyclopropane | Nitrous Oxide | Spinal |
| Nausea Vomiting | 47% | 44% | 29.6% | 17.6% | 15.3% |

### GENITO-URINARY

| | | | | | |
|---|---|---|---|---|---|
| | Spinal | Cyclopropane | Nitrous Oxide | Ethylene | Ether |
| Retention | 22% | 11.1% | 11.1% | 7.1% | 4.33% |
| | Nitrous Oxide | Ether | Ethylene | Spinal | Cyclopropane |
| Cystitis Nephritis Hematuria | 4% | 4% | 4% | 2% | 2% |

### CENTRAL NERVOUS SYSTEM

| | | | | | |
|---|---|---|---|---|---|
| | Spinal | Cyclopropane | Ether | Ethylene | Nitrous Oxide |
| Headaches | 4% | 2.66% | 2.66% | 2.3% | 1.32% |
| | Cyclopropane | Nitrous Oxide | Ethylene | Ether | Spinal |
| Total Cases No Complications | 168 | 143 | 48 | 107 | 57 |
| Percentage No Complications Total Cases | 56% | 47.7% | 37% | 35.7% | 19% |

*The deaths which occurred in this series of cases are grouped according to cause and the anaesthetic administered*

| | Cyclopropane | Ethylene | Nitrous Oxide | Spinal | Ether |
|---|---|---|---|---|---|
| Toxemia | 3 | 2 | 1 | 8 | 7 |
| Malignancy | 1 | | 2 | 3 | 4 |
| Hemorrhage | | | 3 | 2 | 2 |
| Shock | | 1 | 3 | 1 | 1 |
| Embolus or Thrombus | 2 | 1 | 1 | 1 | 2 |
| Other Circulatory Diseases | | 1 | 1 | | 1 |
| Pneumonia | 2 | | 1 | | |
| Other Respiratory Causes | | 1 | 1 | 5 | |
| Total | 8 | 6 | 13 | 20 | 17 |

Time of Occurrence—

| | Cyclopropane | Ethylene | Nitrous Oxide | Spinal | Ether |
|---|---|---|---|---|---|
| First 12 hours | 1 | | 2 | | 3 |
| First 24 hours | 1 | | 3 | 2 | 2 |
| First 48 hours | | 2 | 1 | | 1 |
| First 72 hours | | | 1 | 2 | 1 |
| Later | 6 | 4 | 6 | 12 | 10 |

Of the deaths occurring in those cases which had had cyclopropane, none could be attributed to the anaesthetic.

There were two cases of pneumonia, both hypostatic in type—one occurring five days post-operative in a diabetic, and the other thirteen days post-operatively in a patient with carcinoma of the stomach.

# Rheumatic Heart Disease in Children*

HOMER A. RUPRECHT, M.D.
TULSA

When the pediatrician thinks of heart disease he thinks first and foremost of rheumatism. This disease contributes from eighty to ninety per cent of all the organic heart ailments of childhood. It must be thought of, not as a disease of the heart, but as a generalized systemic infection, chronic in nature, characterized by periods of quiescence and exacerbations, but exerting its most serious and ravaging effects on the heart.

## INCIDENCE

Rheumatism, at least as far as the initial attack is concerned, is essentially a disease of childhood, and therefore to the pediatrician belongs the chief responsibility in caring for the patient during that all-important stage when the heart may be protected or damaged depending upon the care it receives. The age incidence curve of rheumatic fever begins in the intra-uterine period. Several cases have been reported where babies born of rheumatic mothers, have had rheumatic heart disease at birth. The most authentic of these was reported by Kissane and Koons.[1] In their case the mother had had attacks of recurrent rheumatic fever since the age of twelve. At the age of twenty she had been told that she had rheumatic heart disease. At the age of twenty-five she had rheumatic fever throughout her second pregnancy and the child was born with red, swollen, painful (on motion) joints. Abnormal sounds were heard over the heart immediately after birth and the child was noticeably short of breath. However, he lived until the age of nine. An autopsy revealed a severe pancarditis with involvement of all the valves and a fully developed button-hole mitral stenosis. However, such experiences are rare and only three per cent[2] of rheumatic childhood infections occur before the age of two. The curve rises gradually until the age of three, and then sharply, reaching a maximum at the age of ten, after which it declines rapidly until the post-pubertal period is reached. The period of greatest incidence is from six to twelve.

The incidence seems to be about equal in the two sexes.

Most observers agree that there is a definite familial tendency to the disease but whether this is based on the inheritance of a susceptible constitution, to similar environmental factors, or to contagion, is not clear.

The disease affects all social classes but is more prevalent in the lower economic strata. This difference is apparently more marked in England than it is in this country. Paul *et al*[3] in a study of school children in New Haven, Connecticut, found that it was fifty per cent more prevalent in the children in the poorer school districts than among the better. In the "poor" public school districts the incidence was forty-eight per thousand; in the "better" public school districts the rate was thirty-two per thousand; while in the small private schools the rate was only six per thousand. In a similar study of 7,914 undergraduate students at Yale University Paul and Leddy[4] found that the incidence was 8.2 per thousand contrasted with fifteen per thousand which is the average obtained from statistics of comparable age groups of individuals in other walks of life. They also found that of the Yale students the incidence was 12.5 per thousand for high school graduates in contrast to 5.8 per thousand for private school graduates.

Figures on racial incidence are unreliable because they are confused by geographical and economic differences. However, it seems that the American Negro is less susceptible to rheumatic disease than is the white man.

The geographical incidence of rheumatic fever is of particular interest to many of us in Oklahoma who have attended medi-

*Read before Section on Pediatrics and Obstetrics, Annual Meeting, Oklahoma State Medical Association, Enid, April 8, 1936.

cal school and received our hospital training in the north. Rheumatism prefers the colder temperate climate. Heat and a low humidity are its worst enemies. It is extremely prevalent in England. It is almost absent in some sections of the tropics. It is most common in this country, in New England and along the Great Lakes. It is comparatively uncommon in the South and Southwest. I rarely see a case where the initial infection was contracted in or south of Oklahoma. White of Boston states that approximately thirty-six per cent of organic heart disease is rheumatic. Stone and Vanzant of Galveston give 7.3 per cent. At the Charity Hospital in New Orleans rheumatic heart disease comprises about ten per cent of the organic heart diseases.[5] Thus it would appear that rheumatic heart disease occurs only one-fifth to one-third as frequently in the South as it does in New England.

ETIOLOGY

We have every right to assume that the rheumatic syndrome is an infectious disease and yet bacteriological studies so far have proven very disappointing. It is generally believed that it is a streptococcal infection, and yet we have very little sound scientific evidence to support this contention. Nearly everyone working with these conditions has occasionally isolated streptococci from the blood stream; however, the streptococci vary widely in character and their relationship to the disease is very questionable. Rheumatic exudates are characteristically sterile and tissue sections do not show the presence of bacteria. Allergy has been called upon to explain the local tissue manifestations. European investigators have attempted to relate the disease to tuberculosis and Lowenstein claimed to be able to isolate tubercle bacilli from the blood stream. It has been found that the sera of patients with rheumatic fever show a higher agglutination titer against streptococci than do normal sera. It has been found that during a rheumatic exacerbation streptococci are found in the throats in great abundance. However, this evidence is all inferential rather than direct and the primary etiology still remains unsolved.

Of the predisposing causes we have climate, familial tendencies, repeated upper respiratory infections, and economic status—which in turn covers food, clothing, shelter, medical attention, over-crowding, etc.

Scarlet fever is generally thought to cause heart disease either identical with or indistinguishable from rheumatic heart disease.

SYMPTOMATOLOGY AND PATHOLOGY

*Rheumatism is a constitutional disease* which is important because of its effect on the heart. Kaiser[2] in a study of 1200 rheumatic children found the heart to be involved in sixty-four per cent. In thirty-nine per cent a definte arthritis was present; in thirty-two per cent mild joint pains occurred. Adding these last figures, seventy-one per cent showed some form of joint involvement. Chorea was noted in twenty-nine per cent. Muscular or "growing pains" were found in eighteen per cent. Rheumatic pneumonia occurred in 2.8 per cent, erythema nodosum in 1.9 per cent, rheumatic nodules in 1.3 per cent, rheumatic pleurisy in 1.1 per cent, and purpura in 0.4 per cent. Of the minor symptoms, fever, sore throat and tonsillitis, fatigue, anorexia, pallor, epistaxis, headache, abdominal pain, heart pain, and nausea are the more important.

The heart involvement is usually a pancarditis—that is, the endocardium, the myocardium, and the pericardium are all involved. The younger the patient the more severe and generalized the cardiac process is apt to be. One or all valves may be involved. The mitral valve is most commonly affected, the aortic next, and the right sided valves with considerably less frequency. The lesions usually present both stenosis and insufficiency, although the stenosis is usually not apt to be recognized clinically until the process is relatively advanced. In mitral disease a systolic murmur develops early, frequently replaces the first sound, and is heard best at the apex. As the stenosis becomes advanced a diastolic and presystolic murmur appears. In aortic disease the diastolic murmur is heard relatively early because the insufficiency is the earliest functional development and it is only when the process is advanced that the systolic thrill and the murmur of aortic stenosis appear. A pulsating liver, jaundice, cyanosis, and at times distended and pulsating neck veins

are indicative of tricuspid disease. The pericarditis may be dry or with an effusion. A pericardial friction rub may be present in either variety. An effusion may have to be tapped if tamponade develops. At times the effusion may be so large as to compress the left lower lobe of the lung and simulate pneumonia. Pericarditis may be followed by Pick's syndrome. The myocarditis may be of any degree of severity and upon it plus the severity of the generalized infection depends the immediate outcome. The mechanical embarrassment due to the distortion of the valves is of importance only as time goes on.

The joint symptoms vary from mild aches and pains to severe multiple arthritis with effusion, pain, swelling, and fever. I have seen cases where nearly every joint in the body was involved and when the slightest motion meant excruciating pain. In the great majority of instances the joints clear up withoue any residue. Occasionally in the chronic or recurrent form it seems to gradually merge into a condition indistinguishable from a rheumatoid arthritis.

Chorea is another classical manifestation of the rheumatic syndrome. Some writers regard Sydenham's chorea as always a rheumatic manifestation. Schwarz[6] states that in a series of twenty-three cases of "pure" Sydenham's chorea, unassociated with any other rheumatic state, observed for seven years or longer, heart disease followed in one hundred per cent. Most authors believe that heart involvement is much less commonly associated with chorea than with the arthritis; in fact, some believe that not all cases of chorea are due to rheumatism. However, when heart disease is associated with chorea it is likely to have a slow insidious onset and to be less severe than when it is associated with arthritis.

Rheumatic arthritis and chorea are the classical examples, the things we find in text books; if present they make the diagnosis very simple. Unfortunately, and this is especially true in the southern portion of this country, we are often forced to rely on less definite findings. We may have a vaguely ill child, not very sick and yet not well, perhaps a low grade fever which may easily be missed if the temperature has not been taken at regular intervals, he is somewhat pale and listless, the appetite is poor, there is some headache, vague abdominal pain possibly with some nausea and vomiting, and he has lost weight or possibly only failed to gain normally. Such a train of symptoms should make us suspect rheumatism as a possible cause. Tuberculosis is often erroneously diagnosed. Fever of unknown origin lasting more than a few days more often means rheumatism than any other single disease.

It is at this point that the systolic murmur becomes important. As soon as we suspect rheumatic disease we listen carefully for an apical systolic murmur and if we hear one we are usually convinced that the child has heart disease, although we know, or should know, that many children have such a murmur or will develop one in the presence of fever or a nervous tachycardia. Untold numbers of people have been consigned to a life of semi-invalidism unnecessarily because of a functional systolic murmur. A good general rule is never to make a diagnosis of heart disease because of a systolic murmur if one were unable to make the diagnosis if the murmur was not there. There are exceptions, of course. Experience will aid one to recognize a murmur that has an "organic" sound to it. Often one can watch the development of a murmur disassociated from other factors which would tend to bring out or increase a functional murmur.

The electrocardiogram is of importance in that it often gives us the first evidence of cardiac involvement. The changes shown cover practically the entire range of electrocardiographic abnormalities. Some of the more common findings are sinus tachycardia, S-A block, premature contractions, auricular fibrillation, slurring of QRS complex, A-V block, bundle branch block, and changes in axis deviation.[7 8]

## MANAGEMENT

In the treatment of rheumatic disease we are concerned almost exclusively with the actual or potential danger to the heart. For purposes of treatment rheumatism may conveniently be divided into three phases: (1) Rheumatic infection be-

fore the heart has been involved, (2) active rheumatic infection of the heart, and (3) permanent impairment of cardiac function due to cicatricial changes.

In the presence of rheumatic infection but without heart involvement we are concerned primarily in the prevention of cardiac disease. Here rest is of prime importance. It should be absolute bed rest. It should continue for at least two weeks after all evidences of activity have disappeared. Then the resumption of activity should be very gradual and the patient kept under the closest observation for any recrudescence. What are our criteria for quiescence or "arrest"? The temperature must be normal. The leucocyte count must be within normal limits. However, infection may be present even if the white count is normal. The neutrophil percentage and the Schilling count are more delicate and therefore more helpful. Serial counts are of more value than single ones. The sedimentation rate is perhaps the most useful single laboratory procedure. Obviously all subjective and objective manifestations must have disappeared. Special symptoms must be treated as they arise. Salicylates are extremely useful in controlling the joint signs and fever. Restlessness and irritability must be controlled with sedatives. Sedatives must be used to control the symptoms of chorea. The patient should have a normal, well-balanced diet with an adequate vitamin intake.[9] Obvious foci of infection, especially about the upper respiratory passages, should be attended to. However, this does not mean the indiscriminate removal of tonsils.[10 11] Kaiser concludes that the initial attack of rheumatic infection occurs one-third less frequently in tonsillectomized children than in the control group. On the other hand, once a child has become infected with the rheumatic virus, tonsillectomy does not lessen recurrences nor mitigate the severity of the process. Perhaps next in importance to rest is the removal of the patient, when possible, to a warm, dry climate.

If an active infection of the heart is already present the above outlined treatment must be even more rigorously carried out. The period of rest must be extended—usually to at least six months, and often more as circumstances demand. Here the pulse rate is an added factor that must be carefully watched, and as long as the rate remains above normal it must be assumed that infection is still present. Even after the resting pulse rate has returned to normal its response to exertion must also be within normal limits. Treatment with a streptococcus vaccine was tried with some favorable results.[12 13 14] This led to the use of typhoid vaccine for non-specific therapy. More recently fever therapy has been added as an apparently valuable aid in the treatment of rheumatism both with and without carditis. Digitalis at this stage is of little value unless fibrillation is present.

In the third stage we are dealing with fully developed heart disease the sequelae of the active infection. In the endocardium we have stenoses and insufficiencies, in the myocardium we have scar tissue between and even replacing the muscle fibers, in the pericardium we have adhesions—all these are scars which follow the healing process of the preceding acute infection. The treatment is that of heart disease in general. The majority of this group are seen by the internist rather than the pediatrician.

## COURSE AND PROGNOSIS

The initial infection of the rheumatic state occurs principally in childhood. About one-half of one per cent of the school population are affected by it. Of this number sixty-four per cent develop heart disease. The average age at which the primary rheumatic infection was acquired is seven years. According to Stroud at the end of ten years forty per cent are either dead or totally disabled.[15] Kaiser,[2] however, found that only 8.2 per cent were dead at the end of ten years. Davis and Weiss[16] state that five per cent of all deaths due to rheumatic heart disease occur in the 0-9 age period, and twenty per cent in the 10-19 age period. Morse,[17] in a follow-up study of one hundred children seen during their first attack of "endocarditis" ten to thirty years previously, found that thirty-six were dead, three were cardiac invalids, and sixty-one were normal, alive and well. Of the sixty-one, thirty-seven had apparently normal hearts.

He stated that in a large proportion of the fatal cases, death occurred early.

## SUMMARY

1. Rheumatic fever is a chronic infectious disease of unknown etiology most prevalent in the colder temperate climate, occurring most frequently among the lower economic class, and producing its most serious effects upon the heart.
2. The relationship between rheumatic fever and the streptococci is highly suggestive but still unproven.
3. Prophylactic measures are limited to general hygienic measures, change of climate, and possibly tonsillectomy if done prior to the onset of the disease.
4. Actual treatment still remains largely symptomatic and rest is the most important factor of all. Fever therapy is a promising addition to the older methods of treatment.
5. The mortality rate is still very high.

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### Congenital Bony Temporomandibular Ankylosis and Facial Hemiatrophy

Lester W. Burket, New Haven, Conn. (Journal A. M. A., May 16, 1936), believes that a bony ankylosis of the temporomandibular joint occurring in the new-born is of sufficient rarity to warrant a report. The patient, a full term spontaneously delivered male infant, showed true congenital bony ankylosis in the region of the left temporomandibular joint and alveolar ridges associated with maxiliary and mandibular hemiatrophy. Labor was not abnormal, but a moderate hydramnios was present. Undoubted evidence of a typical Horner's syndrome was observed. Lysis of the fibrous and bony fixations was performed twenty-four hours after birth, which permitted fairly normal mandibular movements. Bony ankylosis recurred in two months; however, a sufficient anterior aperture remained to permit adequate feeding. It is possible that reankylosis could have been prevented by the insertion of a fascial or muscular slip between the bony fragments. Ankylosis occurring in the first few years of life, provided sufficient nourishment can be obtained, is best treated after six years of age, at which time the cooperation of the patient, so essential to a satisfactory functional result, can be perhaps secured. Moreover, the first permanent molars are usually erupted at this time, enabling the placing of an orthodontic appliance to stimulate development of the small mandible. Reeducation of speech may be necessary, but this is usually accomplished within two months after operation, provided no other abnormality is present.

### Clinical Observations With Insulin Protamine Compound

Randall G. Sprague, Benjamin B. Blum, A. E. Osterberg, Edwin J. Kepler and Russell M. Wilder, Rochester, Minn. (Journal A. M. A., May 16, 1936), find that the immediate effect of insulin-P (insulin protamine compound) is much less than that of insulin-R (regular insulin). When the former is used alone, and given as a single dose before breakfast, the meals of the first few days provoke glycosuria, but when the dose is properly adjusted the level of the blood sugar on successive mornings decreases progressively and the elevating effect of meals diminishes until, by the end of from four to six days, a normal level of blood sugar may be attained even in cases of severest diabetes. Supplementing insulin-P with small doses of insulin-R will shorten the period of containing control. Insulin-R should not be mixed in the same syringe or injected into the same site with insulin-P. It has not been necessary to continue the supplementary use of insulin-R after the first few days in the milder cases, but probably in many cases such supplementary use of insulin-R will be desirable, especially in emergencies. Until more experience has been obtained it would appear that insulin-R will be the insulin of choice when quick action is desirable, as in the treatment of acidosis. Although insulin-P, in many cases, makes possible effective management of diabetes with only one administration of insulin a day, and with less insistence on rigid control of the diet, its careless use or disregard of the diet is attended with danger.

### Glossodynia

According to Sherman F. Gilpin, Rochester, Minn. (Journal A. M. A., May 16,1936), a search of the records of the Mayo Clinic reveals only forty-eight cases of glossodynia. An attempt has been made to determine, if possible, some of the factors in its etiology and the means by which some patients have obtained relief, as well as any other information pertinent to the problem. As others have found, the majority of the sufferers (thirty-seven) were women. The average age of the whole group was 54.3 years. The ages of the male patients varied between twenty-seven and seventy-three years, and the ages of the female patients varied between twenty and seventy-two years. Only ten of the patients gave a history of migraine. Only five patients could reasonably be classified as being depressed; nevertheless, the incidence of mental depression is higher among those who suffer from glossodynia than it is among the general population. In eleven cases there was evidence of arteriosclerosis of the central nervous system. Here again, therefore, is another possible factor in the production of glossodynia. In the sixteen cases in which gastric analysis was performed, the values for the gastric acidity were normal. In five other cases the values for the free hydrochloric acid in the stomach varied from eight to sixteen. Anacidity was present in only three cases, and in only one of these cases was there any anemia whatever. Cancerophobia has been indicted by one writer as the cause of glossodynia. Only eight patients expressed this fear, which in some cases amounted to a phobia, and it is difficult to determine whether this fear or the burning came first; a few seemingly had acquired the burning first. Most patients whose tongue, palate or mucous membranes are the seat of a burning discomfort have, of course, had their teeth examined. As so often happens in other conditions, doctors and dentists advise extraction rather frequently on empirical grounds in these cases. Consequently, it was not surprising that of the forty-eight patients in this series twenty had lost all or many of their teeth. Thirty-seven of the forty-eight patients complained of various functional disorders, in addition to the glossodynia. It is assumed and maintained, although with not too great certainty, that thirty-seven of the group were psychoneurotic individuals whose many and varied symptoms classified them as such from the outset. These other neurotic symptoms included burning of the legs, continuous headache, a drawing sensation from the stomach to the rectum, continuous pain in the cheek, generalized aching of long duration, burning in the vagina and rectum, a feeling as of a lump in the throat, discomfort in one apparently normal tonsil, and the taste of pus in the throat. Gastro-intestinal symptoms, presumably functional, also should be mentioned with this group. Perhaps one of the best evidences of the fact that glossodynia is a psychoneurotic manifestation is the way in which some of the patients report they received relief from their distress following psychotherapy. Through subsequent visits to the clinic or letters of inquiry, it was possible to learn further about the course of the disease in twenty-five cases. Of the twenty-five cases, complete recovery was reported in eight. Five other patients felt that they were somewhat better than they had been. Three others had found that they had discovered the cause of their discomfort. In nine of the twenty-five the condition was either worse or not better. In none of these cases were there found any local or general positive physical manifestations except for an occasional bad tooth and achlorhydria, which was present in three cases. The burning in this series involved the tongue and the mucous membrane of the mouth, cheek, lips, pharynx, palate and even of the nose. The discomfort included not only burning but such sensations as a metallic or bitter taste in the mouth, prickling, sticking, an acid taste, and a feeling as if a hot iron had been placed on the part affected.

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### Twenty-five-Year Old Error in Measuring a Giant

Charles D. Humberd, Barnard, Mo. (Journal A. M. A., May 16, 1936), is convinced that Cushing's measurements of John Turner (8 feet 3 inches) was in error by at least one foot, perhaps more, for the reasons which follow. Cushing's text presents two good full length photographs of the nude and barefooted giant. An inspection shows that this patient's elongation was quite apparently that of the infantilism-eunuchoid, nonacromegalic type. To one who has studied circus giants and applied a tapeline to them it is evident from these photographs that Turner, though he was huge, was far short of eight feet in height. In the write up of the physical examination the doctor states that "his height is estimated at about eight feet." It is known that the eye always overestimates the exact height of giants—exhibition giants take advantage of the public's credulity, for it is axiomatic that no giant (or no dwarf) ever told the truth about his dimensions! The majority of them, too, customarily do a bit of tinseled falsifying anent the number and the size of their immediate relatives. When the photograph of Turner is studied carefully it will be noted that the giant is stooped, though his head is erect. If a pair of dividers is used to take off the dimensions in this photograph, and the points of the instruments are then measured against a scale, the result will be approximately seven feet three inches. If a man of eight feet three inches bows himself over until his height is lowered to six feet five inches, as is Turner in one of Cushing's photographs of him, he would appear very much more stooped than the photograph shows. A quick test of such a stoop will show at once that Turner has not bent himself to any such extent, for it puts one's spine quite nearly horizontal.

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### Self-Performed Operations

John G. Frost and Chester C. Guy, Chicago (Journal A. M. A., May 16, 1936), review the recorded cases of autosurgery which fall into one of four major groups: (1) surgeons who have operated on themselves, (2) normal minded individuals who have been forced by severe pain or in the absence of medical attention to perform autosurgery, (3) the sexual perverts and those suffering from an acute psychosis, intense sexual excitement, or anger resulting in self-inflicted mutilations or amputations, usually of the genitalia and (4) those who, because of utter ignorance or feeblemindedness, have attempted the surgical correction of some obvious disease or abnormality. A case (group 4) of major autosurgery, twice performed with eventual recovery is reported. The patient's self-performed operations were undertaken for what seemed, to his simple mind, to be good reasons, castration and the resection of a loop of jejunum 11.5 cm. in length, the former was performed without any medical attention and the latter with surgical anastomosis eleven hours after the injury. Comments and diagnoses by the attending staff of the Psychopathic Hospital on the patient were that he was a mental defective but not committable.

# THE JOURNAL
OF THE
## Oklahoma State Medical Association

Issued Monthly at McAlester, Oklahoma, under direction of the Council.

VOL. XXIX JUNE, 1936 Number 6

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Entered at the Post Office at McAlester, Oklahoma, as second-class matter under the act of March 3rd, 1879

This is the official Journal of the Oklahoma State Medical Association. All communications should be addressed to The Journal of the Oklahoma State Medical Association, 203 Ainsworth Building, McAlester, Oklahoma. $4.00 per year; 40c per copy.

The editorial department is not responsible for the opinions expressed in the original articles of contributors.

Reprints of original articles will be suplied at actual cost provided requests for them is attached to manuscripts or made in sufficient time before publication.

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## *EDITORIAL*

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The above appears to be sufficient reason for patronizing our advertisers and it might be well to carry out the suggestion that you ask the "detail" man if his product is Council Approved.

### ANNUAL MEETING OF THE AMERICAN MEDICAL ASSOCIATION

Your Editor has just returned from Kansas City where he attended the Annual Meeting of the American Medical Association and with seven thousand other doctors enjoyed a most pleasant and profitable meeting. Kansas City hotel facilities were practically exhausted; however, everyone appeared to be comfortably cared for.

This meeting was the first to be held in the new Kansas City Auditorium where

every facility was present for the technical and scientific exhibits as well as for the meetings of the various sections.

It was with deep regret that the members of the American Medical Association heard of the serious condition of Dr. J. Tate Mason and his inability to be present at his inauguration. However, he was inaugurated though *absent pour cause de serieuse indisposition*, and this ceremony paid to Dr. Mason the tribute which the House of Delegates had elected him to receive a year ago, even though a note of sadness prevailed.

Many prominent doctors and laymen appeared upon the general program but the one probably most enthusiastically received was Governor Alf Landon of Kansas, who in his address thoroughly endorsed the attitude of the American Medical Association toward regimentation in medicine.

You will of course receive, through the columns of the Journal, the report of your Delegates who were very attentive at all meetings of the House of Delegates and actively participated in its deliberations.

In all, the 1936 meeting of the A. M. A. rivaled all preceding meetings in attendance and interest.

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## *Editorial Notes—Personal and General*

### NINE APPOINTMENTS MADE

Nine new appointments were made to the clinical staff of University Hospital. They include Dr. Emil G. Reed, Dr. George Kimball, Dr. Jesse D. Herrmann, Dr. Clifford Fulton, Dr. John Powers Wolff and Dr. Gregory Stanbro, assistant in surgery; Dr. LeRoy D. Long, Jr., associate in surgery, and Dr. Robert L. Noel and Dr. Howard Shrobe, instructors in orthopedic surgery.

Dr. Joseph B. Goldsmith was promoted from assistant to associate professor of histology and embryology on the medical school staff.

On the recommendation of Dr. Robert U. Patterson, dean of the school, the regents voted to limit the age of members of the hospital staff to sixty-five years, effective in 1937.

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DR. and MRS. W. J. WHITAKER, Pryor, are visiting in New Orleans, where Dr. Whitaker will attend the Turo Infirmary.

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DR. J. C. BUSHYHEAD, Claremore, is reported much improved after suffering a light stroke a few days ago.

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DR. W. ALBERT COOK, Tulsa, gave a paper on EYE INJURIES at a meeting of the Creek County Medical Society, May 7th.

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DR. J. B. CARMICHAEL, Duncan, has been appointed as County Health Superintendent of Stephens County, effective May 26, to fill the vacancy caused by Dr. D. Long's death.

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## *News of the County Medical Societies*

On May 27th, Dr. J. A. Walker, Councilor for the Seventh District called a meeting of the societies in his district at Shawnee. Dinner was served at the Aldridge Hotel after which proposed legislation was discussed by Drs. Speed, Willour, and Geo. R. Osborn.

Following these addresses the forty members present entered into general discussion of our legislative problems from which much good was derived and many pertinent suggestions made.

It was reported at this time that several counties throughout the state had made definite arrangements to meet their quota of the allotment for expense of the legislative program.

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## *RESOLUTIONS*

### DOCTOR D. LONG

At a called meeting of the Council of the Oklahoma State Medical Association May 24, 1936, the following resolution was presented and unanimously adopted:

"WHEREAS, this Council has received the sad information of the death of Dr. D. Long of Duncan, Oklahoma, and

"WHEREAS, Dr. Long, for many years, has been a member of this Board of the State Medical Association and rendered through it and in many other ways most valuable service to organized medicine in Oklahoma, and

"WHEREAS, he has been recognized among the doctors of the state as one of the most beloved members of the medical profession,

"THEREFORE BE IT RESOLVED, that we receive this information with deep sorrow, realizing our loss in no longer having the benefit of his counsel and advice, and

"BE IT RESOLVED, that we extend to the family our deepest sympathy and assure them of our sincere desire to share with them this burden of loss, and

"BE IT FURTHER RESOLVED, that a copy of this resolution be made a part of the minutes of this meeting, that it be published in the Journal of the Oklahoma State Medical Association, and that a copy be sent to the family and to the press of his home city."

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### DOCTOR J. P. BISHOP

WHEREAS, it is with deep regret that we learn of the death of our friend and fellow in medicine, Dr. J. P. Bishop, of Aline, Oklahoma, who has recently gone to his reward, and

WHEREAS, the Woods-Alfalfa County Medical Society has lost a member who has been a faithful attendant and worker for many years, and

WHEREAS, he has practiced in the Aline community for many years and has always been active

in the practice of medicine and strictly adherent to the principles of medical ethics, and

WHEREAS, he will always be remembered as a man of high professional stand, high morals, and square dealing, with a warm heart for the poor and those in need of medical service, therefore

BE IT RESOLVED, that the Woods-Alfalfa County Medical Society in its regular session in Cherokee Tuesday, May 26, 1936, expresses its sincere regrets in the loss of its fellow worker and friend and extends its heartfelt sympathy to his bereaved family, and

FURTHER BE IT RESOLVED, that a copy of these resolutions be sent to the bereaved family and a copy be tendered the local paper, and the Journal of the State Medical Association.

L. T. Lancaster,
M. G. Dunnington,
O. E. Templin,
Committee.

**Popliteal Aneurysm**

Arthur H. Wells, Duluth, Minn.; Clay E. Coburn and M. A. Walker, Kansas City, Kan. (Journal A. M. A., Aprill 11, 1936), believe that it is not generally known that, with the exception of aneurysms of the aorta, popliteal aneurysms are found more frequently than those of any other artery. According to Matas, they are by far the most frequent of the surgical and operable types of the disease. Because of this frequency, their characteristic rapid progression and the efficacy of proper surgical intervention, the authors give a brief review of the subject and present a case illustrating tragic characteristics of the disease. The relative frequency of popliteal aneurysms, their often rapid progresion, the disastrous gangrene in unaided cases and the efficacy of proper management makes this an important subject for every physician who is likely to encounter the condition.

# OBITUARIES

## DOCTOR HOWARD ALONZO WAGNER

Howard Alonzo Wagner was born at Bath, Pa., October 4, 1875, and died at his home "The Oaks" in Shawnee, Oklahoma, on April 26, 1936, of coronary thrombosis. Dr. Wagner was a bachelor. He leaves seven brothers, all living near Bath, Pa.

He was graduated from the University of the South at Sewanee, Tenn., with the degree of Doctor of Medicine in 1901 and came to Shawnee, Okla., immediately after graduation and had practiced continuously in this city until his death, doing a general practice, with special attention to urology.

Early in the War he volunteered and spent twenty-one months with the American Expeditionary Forces in France and Germany. He was discharged as a Major in the Medical Corps.

He was a member of the Pottawatomie County Medical Society, the Oklahoma State Medical Association, and American Medical Association. He was a member of Shawnee Lodge No. 107 A. F. & A. M., Order of the Eastern Star, Indian Consistory at McAlester, Okla., India Temple of Oklahoma City, and was a thirty-third degree honorary Mason. He held many high offices in the various Masonic bodies. He was also a member of the Elks lodge and the American Legion.

Dr. Wagner devoted his time, his energies, and his funds to the youth of the city and state, helping many young men through school and college. His heart was open to all worthy youths who wanted an education. For twenty-four years he had been interested in the Masonic Home for Orphans at Guthrie, Okla., bringing cheer and happiness with his annual Easter party to the children there. The DeMolay, the high school athletic teams, and the various organizations of young men in the city were all his boys and he helped them in every way. His home, "The Oaks," was open to any worthy organization for their parties and picnics and was a second home to all his friends. Although a bachelor, he probably had the largest "family" in the state as every one who knew him loved and respected him.

Interment was in the Shawnee Mausoleum, Fairview Cemetery, Shawnee, Okla.

## DOCTOR JAMES CLINTON HAWKINS

Dr. J. C. Hawkins, fifty-two-year old Blackwell physician, died May 15th, following an automobile accident in Missouri, when he swerved his car from the road to avoid hitting a truck. Mrs. Hawkins was seriously injured but will recover.

Dr. Hawkins was born at Brumley, Mo., February 18, 1884, and was a graduate of the Washington University Medical School at St. Louis. He came to Blackwell in 1917 where he has practiced until the time of his death. He served as president of Kay County Medical Society, Blackwell Rotary Club, and Blackwell Country Club. Served in the World War and was a lieutenant-colonel in the Medical Reserve Corps at the time of his death.

Dr. and Mrs. Hawkins were returning from the meeting of the American Medical Association in Kansas City when the accident occurred.

Dr. Hawkins is survived by his wife and several brothers and sisters.

Interment was in Blackwell.

## ADDITIONAL DEATHS

(Insufficient data available for obituary.)

Dr. Joseph Patterson Bishop, Aline, Oklahoma, May 13, 1936.

Dr. Reuben Taylor Harrod, Oktaha, Oklahoma, May 19, 1936.

Dr. Dock Long, Duncan, Oklahoma, May 21, 1936.

Dr. John Sinclair Miller, Hugo, Oklahoma, May 4, 1936.

Dr. John W. Sosbee, Stroud, Oklahoma, May 20, 1936.

# ABSTRACTS : REVIEWS : COMMENTS *and* CORRESPONDENCE

## *EYE, EAR, NOSE AND THROAT*

Edited by Marvin D. Henley, M.D.
911 Medical Arts Building, Tulsa

**Contact Glasses, the Invisible Spectacles. Joseph Dallos, M.D., Budapest, Hungary. Archives of Ophthalmology, April, 1936.**

In 1827, Herschel, the English physicist, explained the theory of the "invisible" eye glasses and approximately fifty years later three German scientists also advocated their usage.

To obtain a clear vision the eyeball must be healthy. Astigmatism, congenital or resulting from an operation may be corrected with the invisible eyeglass. Poor sight, caused by injuries or scarred healings of diseased corneas cannot be aided by regularly ground eyeglasses. The cornea cannot be perfectly smoothed and its removal and the insertion of a new one is very hazardous. Experiments of Fisk, in collaboration with the late Professor Abbe, are described and the results considered favorable.

August Muller, a young medical student was not so optimistic. He discussed the superiority of the curved lenses over the simply ground ones, which fact is considered in that light today. The disadvantages of securing perfect vision with spectacles is discussed, including inability to participate in sports, necessity of becoming accustomed to exceedingly strong lenses, the distortion of objects, etc.

About this time a third German, F. A. Muller, maker of artificial eyes, made a protective shell for a sixty-year old man, blind in one eye, and whose eyelids of the other eye were destroyed by cancer. In place of the painted iris, Muller blew a center that was thin and transparent and the patient wore this for over twenty years. Consequently it became known and more shells were made—and these more perfect—to the extent that the patients who followed were able to wear them all day without discomfort.

However, ophthalmologists for various reasons did not accept the invisible eyeglasses as the approved method in aiding impaired vision.

Until 1930 the idea was carried out that the shells could not be worn except in special cases, due to limited optical correction and that ground glasses were the reverse, and could not be prescribed for practical use.

Many tried to combine the advantages of the two but this was impossible, so it became necessary to perfect the ground shells, which was undertaken by the Zeiss factory.

Since 1930 many experiments have been carried out with the Zeiss type of ground contact glasses. The results have been highly praised by the laity as well as the ophthalmologist, although the latter should not wholly trust the patients word but should depend on further proof.

It necessitates a very great number of bowls to find a perfect fit and even in the University of Kiel where there are over three hundred types ($10,000.00), only a small percentage can be fitted.

The difficulty in giving satisfaction is easily understood. The eyeball must not be injured and it must be taken into consideration that it is not a perfect sphere.

In 1932 the author found Poller's negocoll, a material out of which a cast of the eye could be made. This takes only a few seconds and is not particularly painful. Further successful experiments were made by the Ophthalmologic Clinic No. I.

At the present time shells are not selected at random but are fitted with great care and precision. The lenses can be worn all day with perhaps a short rest at midday and then removed at night. The removal or insertion is comparatively easy, the care of the shell is simple and replacement, if broken, is easily made.

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**Thyroxine in the Treatment of Otosclerosis. Dr. Max Goldstein, St. Louis. The Laryngoscope, February, 1936.**

Reference is made to the paper of Dr. Albert A. Gray, which was presented to the Section of Otology of the Royal Society of Medicine, on May 3, 1935. The paper was entitled "Treatment of Otosclerosis and Similar Types of Deafness by the Local Application of Thyroxine." Fourteen cases were reported, seven of which showed quite a degree of improvement. Improvement consisted of reduction of tinnitus, recurrence of cerumen secretion and actual improvement in hearing.

The same summer Dr. Goldstein visited Dr. Gray at the Ferens Institute, Middlesex Hospital, London, and saw him apply the technique described, in three cases. Although the technique is carefully described in Dr. Gray's publication the author thinks emphasis should be laid on such points as the anaesthesia and puncture of the ear drum, the preparation of the tabloid for injection through the ear drum, and the measurement of sound capacity before and after treatment.

Dr. Goldstein remarks that in his forty years of otologic practice he has never succeeded in securing a complete and safe anaestehsia of the drum membrane. He recounts that in using cocaine in aniline oil once to relieve pain in a case of acute otitis, aniline poisoning followed. Dr. Gray uses for his anaesthesia ten per cent cocaine in aniline oil, but five minutes after instillation, he wipes the canal dry with pledgets of cotton and so removes the possibility of aniline poisoning. The fineness of the pulverization of the tabloid thyroxine, which is held in suspension by mixing with four mm. of distilled water on a sterile watch glass, is one of the essential points in the success of the injection through a fine calibered needle into the middle ear. The point at which the drum membrane is punctured should be half way between the end of the malleus handle and the periphery of the drum membrane in the posterior inferior quadrant. The point of the needle should not touch the bony lip of the round window. In order that the injection

fluid does not escape by way of the Eustachian tube, the patient's head is bent far backward and the mouth opened for about five minutes immediately after the injection.

Difficulty is noted in regard to securing in the United States the tabloid thyroxine which is used by Gray. In making sound estimates before and after treatment, Gray uses the tuning forks, the watch, and conversational and whispered voice. The author uses in addition, the audiometer. He reports four selected cases which tend to confirm the findings and results as obtained by Gray.

Otosclerotics under forty years of age whose hearing is not so deteriorated that comparative tests are difficult, offer the best field for favorable results, according to Gray. If the hearing is improved, it appears to be permanent. If the treatment is a failure, then no harm has been done. It is not a disabling procedure.

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**On the Genesis of Glaucoma. Barkan, Boyle and Maisler, San Francisco. The American Journal of Ophthalmology, March, 1936.**

One hundred cases of glaucoma have been investigated carefully by the authors in the past two years. The examination of the angle of the anterior chamber in the living has not been possible until quite recently. Trantas, in 1907, using an ophthalmoscope with a strong inset convex lens, and making pressure at the limbus outside the point under observation, was the first to report observation of the angle. The advent of contact glass made this research much more accurate. This deflects the incoming and outgoing rays in such a manner that you are able to obtain with the ophthalmoscope a view of the most distant angle of the anterior chamber.

Fick was the first to use this method. Salzman, in 1914, using the contact glass investigated and reported on glaucoma cases. Koeppe, in 1920, using the binocular stationary microscope and the slitlamp reported on the condition of the trabecula covering Schlemm's canal. Troncoso, in 1925, greatly stimulated research on this subject by devising the gonioscope. Thorburn, in 1927, used a contact glass and a loupe of low magnifications and studied one hundred cases of glaucoma.

The authors point out the defects in these previous methods and describe their method in detail. They used a Zeiss binocular corneal microscope and the Vogt arc slitlamp so adjusted that they obtained a highly magnified stereoscopic view of the angle of the anterior chamber throughout its entire circumference. The patient wears contact glasses at the time of the examination and is examined lying down. They found from their investigations that the angle is not of equal width throughout its circumference as has been formerly thought, but narrower above than it is below.

A frequent if not constant finding in the so-called primary glaucoma is the sclerosis and pigmentation of the trabecular part of the angle of the wall. Through the trabecula over Schlemm's canal are pigment dust and granules. Some of these cling to the surface and are likened to barnacles on the hull of a ship as they project into the aqueous. Sclerosis of the trabecula and the stopping of the pores with pigment is given as one of the causes of chronic glaucoma. In a patient with one apparently normal eye, with normal fields and tension, some pigmentary block of the trabecula and Schlemm's canal could be demonstrated, which would seem to point to the fact that pigmentary block is primary and the cause rather than secondary and the result of hypertension.

Some of the different types or stages of glaucoma are discussed. In the compensated, simple, chronic non-inflammatory cases of glaucoma they think stoppage of the pores of the trabecula is the immediate and sole cause of the reduced outflow of the intra-ocular fluid. They think this is the first stage of chronic glaucoma. They think it is also the first stage of decompensated chronic, inflammatory glaucoma in which the iris has become adherent to the angle of the wall and produces occlusion of Schlemm's canal. According to their conclusions it is obvious then that the pigment block occurred before the iris adhesion and was the primary factor in hypertension. Another type or stage of glaucoma described is characterized by a narrowing of the entrance to the angle between the anterior-border ring of Schwalbe and the iris root. This is attributed in many cases to the general advance of the iris-lens diaphragm and in some to a ballonling (bombe) of the iris. These iris adhesions prevent the outflow of aqueous into Schlemm's canal. The classification of the primary and secondary glaucomas is discussed.

Causes for increase in violence of the disease are attributed to increase of obstruction of the filtration angle or to factors having their origin in nerves. Further reports are to be expected from these men in the near future.

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**Indications for Tonsillectomy Viewed from the Point of View of Otolaryngology and Internal Medicine. G. Dohlman. Abstracted by H. V. Forster from Acta Oto-Laryngologica, Vol. XX, 3-4, and Published in The Journal of Laryngology and Otology, June, 1935**

The internist sees the patient during his illness and is in a position to observe the development of any complications as they arise, but the otologist who sees the patient in the quiescent stage only hears a description of the case history, and this from the patient himself.

The conception of focal infection has enlarged the field of indications for operations and a certain confusion exists as to the desirability of doing them.

When we carry out tonsillectomy because of repeated acute attacks or for complications in the joints, heart, kidneys, etc., which have followed these attacks of acute tonsillitis, we have definite theoretical and practical indications for interference; and when we examine such tonsils microscopically we find certain signs suggestive of inflammatory change, but we can hardly say what anatomico-pathological state will justify a diagnosis of chronic tonsillitis or indicate that localized complications or a general toxic absorption would take place.

As there is, on microscopical examination, nothing to distinguish with certainty the tonsil of a healthy person from that of a patient subject to repeated acute attacks accompanied by repeated attacks of polyarthritis or any other complication, we are forced to draw the following conclusions:

1. It is useless to rely on macroscopical signs to judge the normal or pathological tonsil when the microscope cannot decide, and when deciding on operative indications one should neglect the so-called "clinical signs" of a chronic tonsillitis-plugs, hyperplasia, etc., redness of pillars, gland troubles. These only prove past troubles or a more or less accidental irritative condition.

The writer believes, however, that we ought not to refuse to do a tonsillectomy in cases in which the history indicates clearly a relation between repeated tonsillar affections and their complications, even though we do not discover plugs of detritus in the crypts or some other equivalent

sign, because we should then waste time looking for evidence by inspection alone.

2. The second conclusion is a physiological one which Hellman has established, that there is a battle going on in the tonsils between noxious influences and a defense mechanism. Chronic tonsillitis is a normal state unknown only in those animals brought up in an environment free from bacteria.

Because of the indecisive frontier micro- and macroscopically which separates the normal from the abnormal tonsil this consideration has led to the erroneous supposition that all tonsils are unhealthy organs and ought to be removed. Another useless argument is that the same functions belong to all the other lymphoid tissue of the alimentary tract and we cannot remove it all by surgical methods.

A more legitimate conclusion would be that in the clinically diseased tonsil we have a mode of pathological reaction which is expressed by repeated acute attacks within the organ itself, and by a pathological permeability to bacteria and their toxins which can lead to a series of complications.

As this is the case it would be better to try and discover the cause of these modifications of tonsil reaction to bacteria which live in the mouth and tonsil tissue. These reactions may also depend on a local lesion of the tissues resulting from previous severe attacks of tonsillitis. In such cases we can expect a good result from tonsillectomy, but the alterations in question may also be a manifestation of lack of resistance of the organism in general, and then if we carry out a tonsillectomy the patient ceases to have anginas, but persists, nevertheless, in presenting just as often pharyngitis, rhinitis, bronchitis, etc. Both these categories of patients are no doubt well known to us all.

One could argue that it is not so much poor resistance on the part of the body as the vitability or virulence of the bacteria which are important factors, but if we consider that the sole object of bacteria is not to attack men or animals but to behave according to the nutritive medium on which they find themselves implanted we begin to appreciate the importance of the buccal secretions and the reactionary capacity of the tonsils to bacteria.

In some experiments which the writer carried out he noted, for example, how Gram-positive organisms disappeared from the bacterial flora of the mouth cavity after administration of a diet free from carbohydrates.

When certain favorable results follow the operation of tonsil removal in arthritis or chronic septic conditions there are some factors which should not to be lost sight of. Do the rest in bed and enforced starvation have in themselves a beneficial effect? We have also to remember the presence of two bare operated areas in the throat from which a vaccination process may take place.

In conclusion the writer considers that no otologist would refuse to remove the tonsil when, after a careful examination of the case, it had been decided that this organ was responsible as a source of infection for certain pathological lesions, but cases should not be submitted to him for operation on the slender pretext that certain diffuse symptoms originate from it.

The internist must develop a keen sense of responsibility and not propose a "hit or miss" policy or suggest tonsillectomy just because some other line of treatment has failed, neither should the argument be used that the operation is not a dangerous one. Such an argument would be of little use after a possible accident or fatality.

## *SURGERY AND GYNECOLOGY*

Abstracts, Reviews and Comments from
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**The Control of Morbidity and Mortality Following Pelvic Surgery. (A Review of a Second Series of One Thousand Consecutive Personal Cases.) By Walter T. Dannreuther. Surgery, Gynecology and Obstetrics, May, 1936, Pages 791 to 798.**

In January, 1930, this author made a critical analysis of one thousand consecutive personal operative cases immediately preceding that publication with the following summary and conclusion:

"In a series of one thousand gynecological operative cases, there were complications during convalescence in seventy-nine, a morbidity of 7.9 per cent; nineteen patients died, a mortality of 1.9 per cent.

"Post-operative transfusion should never be necessary except in the presence of ectopic pregnancy, secondary hemorrhage, or sepsis.

"Pre-operative cystoscopy and renal function tests will eliminate many useless pelvic operations.

"Severe anemia, pyorrhea, respiratory affections, a compromised myocardium, arterial hypertension, and impaired metabolism, are indications for postponing operation in elective cases; spinal anesthesia may reduce the hazards in emergency cases.

"Radiation and diathermy are dangerous in the presence of active infection or necrobiosis.

"Meticulous peritonealization and burying all suture knots minimize the likelihood of post-operative intestinal obstruction.

"The incidence of pulmonary complications can be lessened by the invariable employment of a skilled anesthetist.

"Correcting indicanuria and obstinate constipation before operation will practically eliminate post-operative pyelitis.

"Persuading the patient to practice active motion of the extremities throughout convalescence is good insurance against thrombophlebitis and embolism.

"Pronounced tachycardia developing during the first half hour of anesthesia is an indication to terminate the operation as soon as possible.

"Unpreventable surgical shock will develop occasionally in profoundly septic patients.

"Patients with a high metabolic rate should not be subjected to radiation."

Since the publication of this series which he designates as Series A, he has collected another group of one thousand personal cases, identified as Series B, and is now comparing the end results for the purpose of assistance in reducing post-operative morbidity and mortality.

He calls attention to the fact that most gynecological operations are elective rather than emergency and that there is usually ample opportunity for thorough pre-operative preparation of the patient.

He has carefully analyzed both series of cases as to morbidity and to mortality with individual tables as to the various types of morbidity complications. The following are the conclusions he draws:

"1. Having conscientiously scrutinized a previous series of one thousand consecutive personal gynecological operative cases, it has been possible to effect a thirty-four per cent reduction in morbidity and a fifty-seven per cent reduction in mor-

tality. In the second series of one thousand, there was morbidity in fifty-three patients and eight died.

"2. Gentleness in separating bowel adhesions, recognition of endometriosis of the rectovaginal septum, refusal to irradiate any patient suspected of harboring a latent pelvic infection, careful peritonealization of raw surfaces, and the use of sheets of guttapercha tissue to cover surfaces that cannot be peritonealized will obviate fecal fistulas and post-operative intestinal obstruction.

"3. The routine administration of ten per cent carbon dioxide in oxygen just before the completion of the operation will minimize the incidence of pulmonary complications in general, and pneumonia in particular.

"4. Dehiscence of an abdominal wound is probably due to tissue hunger and proteolytic digestion of catgut before fibro-plastic production is sufficient to splint the wound, rather than to defective suture material.

"5. Approximation of the vaginal fibers of the levator ani muscles with kangaroo tendon and a subcuticular suture of the skin margin evidently minimize perineal wound infections.

"6. Keeping the bladder free from residual urine lessens the incidence of post-operative cystitis. Residual urine is more of a menace than aseptic catheterization.

"'. Postponing elective operations until the cardio-vascular system is in the best possible condition, and the frequent practice of active motion of the extremities throughout convalescence will practically eliminate post-operative embolism.

"8. Removing the uterus by applying clamps instead of preliminary ligatures is a safe procedure.

"9. Pre-operative blood transfusions should be utilized in all cases of anemia, debility, or sepsis.

"10. Invasion of the cellular connective tissue of the parametrium for the removal of intraligamentary tumors predisposes to subsequent parametritis.

"11. Glucose should always be administered intravenously, never subcutaneously.

"12. It should be the aim of every pelvic surgeon to limit his post-operative morbdiity and mortality to an irreducible minimum."

COMMENTS: Thsi is a bit of conscientious work by a very able gynecologist. Though this report embodies no new investigative work, such publications are of extreme value because they present crystallized thought upon the fundamental purpose of pelvic operative work.

One finds it easy to endorse practically all of the conclusions of Dr. Dannreuther, differing only on minor points. Wendell Long.

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**The Misuse of Tannic Acid. By Frederic Taylor, M.D. From the Journal of the American Medical Association, April 4, 1936, Pages 1144-45.**

Beginning with the statement that the tannic acid treatment of burns has attained its popularity through its success in extremely severe burns, the author takes the definite position that it should not be employed in more or less superficial burns.

Attention is directed to the fact that when only the superficial layers of the skin are involved regeneration takes place from the remaining germinal layer. If the germinal layer is destroyed regeneration takes place from hair follicles and sebaceous glands. The author asserts that in the employment of tannic acid solutions the cells of the germinal layer and the cells in connection with the hair follicles and sebaceous glands are tanned or "fixed." After the epithelial cells in these structures are tanned or "fixed," they do not regenerate.

The author compares the more or less superficial burn to donor sites of a split skin graft. He says that no one would consider treating such a site with tannic acid, and "yet this situation is quite comparable with the usual burn in which regeneration must also take place from the remaining living epithelial cells of the hair follicles."

The final conclusion is that in the great majority of burns it is not wise to institute any therapy that might destroy surviving epithelial cells. It is suggested that bland dressings of saline solution, dilute solution of sodium hypochlorite or ointment be employed until it can be determined "just which epithelium will survive and which will die."

While the author gives credit to the usefulness of the tannic acid treatment in severe burns, he points out that it is probable that the lowered mortality is very largely due to two other factors: (1) An increased interest on the part of the hospital staff; (2) a full appreciation of the result of shock and the replacement of lost fluids in the body.

LeRoy Long.

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## SOME CASE REPORTS

**Case I—Melanoblastomatosis Arising From Neglected Black Mole. Relentless Course. Death in Thirteen Months.**

A married woman, Mrs. J. A H., multipara, thirty-five years of age, came to the Clinic on February 1, 1935, complaining of: (1) Lump in the left breast, painless, noticed for about three and one-half years, during which time it had gradually increased from the size of a small walnut to the size of an egg. (2) A "birthmark" on the right lower back which had been increasing in size for about six months, and had been bleeding occasionally for longer than three months, during which time a dressing had been worn over it.

The general condition of the patient was good in every respect.

Examination disclosed a firm, irregular, moveable mass about two and one-half inches in diameter above the nipple of the left breast. Clinically, it appeared to be a benign mass.

There was a black, bleeding elevated mass about three-fourths of an inch in diameter just above the posterior iliac crest on the right side. It had all the characteristics of a vicious melanotic growth.

Notwithstanding the good general condition, the husband was advised that the growth on the back represented a most pernicious type of malignancy, and that the prognosis was extremely bad.

On February 4, 1935, a sweeping removal of the growth on the back was done at St. Anthony Hospital by the use of an electric cautery. The pathological report was that it was a melanoblastoma. The mass in the breast was removed at the same time. It was a galactocele, without any evidence of malignancy.

The operative wound of the back healed with extreme slowness, probably due to the widespread destructive action of the cautery. Finally, after about six or seven months, it was healed entirely, and there was never any local recurrence.

In the early part of June, 1935, our attention was called to an enlargement in the right groin. It was slightly tender. There was no discoloration. There had been a boil on the outer side of the right thigh. Notwithstanding the location of the boil, we had the forlorn hope that it might be an adenitis resulting from it, but, at the same time,

we regarded it with grave suspicion. A little later there was an enlargement in the anterior axillary fold, right side. There was no discoloration. It was not tender.

These masses—the one in the right groin and the one in right anterior axillary fold—were removed by radio knife on October 8, 1935. They were rather deeply placed, and when removed were black in color. The pathological report was melanoblastoma, metastatic. The general condition of the patient was good in every respect.

In the early part of January, 1936, there were some nodules about the operative scar in the right groin. At that time the patient felt very well, but a blood count showed slight reduction in hemaglobin and red blood cells.

On January 29, 1936, the scar in the right groin and a considerable amount of tissue on each side of it, extending down to the muscular structures, were removed. The mass containing several black bodies, the larger being about four-fifths of an inch in diameter, had all the gross characteristics of a melanotic growth. The pathological report was melanoblastoma.

The patient did pretty well for about a week, when there was a secondary hemorrhage from the operative wound, and at the same time excessively profuse menstrual discharge. On February 8, 1936, the vagina was packed, and the operative wound in the groin explored. There was a little oozing about the skin margins. All oozing points were controlled by suture-ligatures, and a firm pack put into the wound cavity. There was no additional external hemorrhage of importance.

After these emergency procedures, there was a transfusion of 500 cc. of citrated blood. The patient felt some better, but complained of pain and distention in connection with the abdominal cavity.

There was vomiting, the vomitus containing a considerable quantity of blood. There was fever and great weakness. There were physical evidences of fluid in the abdominal cavity. Notwithstanding the control of hemorrhage from the operative area and from the uterus, there was a profound anemia with a hemaglobin of fifty, and an R. B. C. of 2,830,000 a few days after the transfusion. The coagulation time was five minutes, forty-five seconds, and the bleeding time four and one-half minutes. There was another transfusion of 500 c.c. of citrated blood, followed by some temporary improvement, but vomiting, the vomitus containing blood, continued, and there was bitter complaint of pain about the abdomen which was very greatly distended. An examination of the abdomen revealed shifting dullness.

On February 18, 1936, there was a hemaglobin of thirty-six and an R. B. C. of 2,560,000. There was another transfusion of 500 c.c. of citrated blood, with only transient benefit. On February 21, 1936, the blood count showed only 1,990,000 red blood cells, with an estimated hemaglobin of forty per cent (Talquist). The patient was extremely weak. There were more evidences of fluid in the abdomen, and a mass, interpreted to be an extension downward from the liver, could be felt in the right upper abdomen. There was another transfusion of 500 c.c. of citrated blood, with considerable temporary improvement.

Early on the morning of March 2, 1936, there was sudden collapse, quickly followed by unconsciousness, and the patient died at 1:20 p. m. that day.

An autopsy confined to the abdominal cavity revealed an enormous amount of blood in the peritoneal cavity, probably mixed with ascitic fluid, which could not be differentiated from the blood. There were numerous black nodules about the structures in the pelvis and in the mesentery. The right lobe of the liver was greatly enlarged, and contained numerous black masses. It was our conclusion that the enormous amount of blood in the peritoneal cavity resulted from hemorrhage due to disintegration of blood vessels by the extension of metastatic masses. The cause of death was obviously melanoblastomatosis, resulting in intraperitoneal hemorrhage and exhaustion.

* * *

**Case II—Extensive Sub-Acute Pancreatitis, Associated With Pronounced Pathology Bile Tract Area. Cholecystectomy With Drainage of Cystic Duct. Recovery.**

H. S. G., white man, sixty-four years of age. History of "indigestion" for four years or longer. During most of that time has been incapacitated for active work. Has had a good deal of pain in the "pit of the stomach."

This patient entered St. Anthony Hospital in the service of Dr. B. E. Mulvey April 3, 1936. The night before coming to hospital there was excruciating pain in the epigastrium. According to the history, it had been necessary to give morphine hypodermatically three times within two hours before the pain was relieved.

He entered hospital about one p. m. with a temperature of 103.4, pulse 78, respiration 20. There was a history of pain in the rectum about two and a half months before coming to the hospital, and that was followed by bleeding from the rectum.

Dr. Mulvey had investigated the patient within a week before his admission to the hospital. The Ewald showed a total acidity of thirty-four, an absence of free HCl, an absence of occult blood. The Wassermann was negative. An x-ray examination during the investigation by Dr. Mulvey did not show any evidences of organic disease of the stomach. There was no x-ray evidence of pathology of the intestinal tract. There was no evidence of neoplasm or ulcer in connection with the rectum.

The patient was extremely uncomfortable. He was semi-delirious. He was extremely restless.

There was moderate regidity of the epigastrium, and a more pronounced rigidity in the right upper abdomen. The abdomen, generally was not rigid. There was occasional audible peristalsis. Taking into consideration the history of indigestion over a long period of time, the excruciating pain in the epigastrium, the moderate rigidity of the epigastrium and the fever, and in spite of the Ewald report and the negative x-ray findings, we considered a perforation into the lesser peritoneal cavity as a distinct possiblity.

The next morning after admission the patient was more comfortable. There was vigorous audible peristalsis. Pulse sixty-four. Temperature 98.6. There was a little staining of the sclerae. The following day the eyeballs were a little more yellow and there was a suggestion of jaundice in connection with the skin.

There was an afternoon elevation of temperature from 100 to 101.8. There was a good deal of nausea, and practically complete anorexia. At the same time, there was less rigidity in the epigastrium. There was still some rigidity in the right upper abdomen, and some tenderness under the right rib margin.

On April 7, 1936, there was an immediate direct van den Bergh reaction with 4.5 mg. per cent azobilirubin per 100 cc. of blood. The bleeding time was one and one-half minutes. Coagulation time four minutes forty-five seconds.

On admission the urine was reddish, clear, acid

in reaction, specific gravity 1.015, albumin trace, glucose 4 plus, acetone negative, indican negative, occasional white blood cell. The blood count showed Hgb. 75, R. B. C. 4,800,000, W. B. C. 6,200, polys. 93, lymphs 7. On April 6, 1936 the W. B. C. count was down to 3,800. On April 7, 1936, it was 7,300. It will be observed that there was not a leukocytosis at any time, and that at one time—April 6, 1936—there was a pronounced leucopenia.

The patient had been given glucose intravenously every day, and that may account to a very large extent for the finding of sugar in the urine.

The jaundice became more distinct, but it was never pronounced.

The patient was observed for a week, during which time, what with the administration of glucose intravenously and normal saline solution subcutaneously, there was considerable improvement in the general condition.

Casting up the data collected during this week of observation, we abandoned the hypothesis of a perforating ulcer into the lesser peritoneal cavity, and concluded, finally, that there was an infection of the bile tract area, probably with stones, with at least a partial obstruction of the common duct.

An operation was performed on April 10, 1936. There was a greatly thickened, whitish gallbladder, containing thin, pale bile, and many tiny black calculi, one of them being fixed in the cystic duct. The omentum was adherent to the gallbladder. The pancreas was greatly enlarged throughout its entire extent. The right lobe of the liver was low, heavy and dark, but there were no nodules found in it. The stomach appeared to be normal.

It was very clear that there was an extensive involvement of the pancreas, and since it was so uniformly involved from head to tail, it was concluded that it was probably not due to a neoplasm, but to an inflammatory process.

The gallbladder was dissected from the liver, beginning at the fundus, down to the cystic duct. The duct was divided, and a very small catheter introduced into it, the catheter being fixed by one catgut suture.

On the evening of the day of the operation the temperature was 100. On the day following the operation there was a gradual elevation of the temperature until it reached 103.4, and it hung around 103 for about twenty-four hours. At that time there was a rather rapid drop to 99, after which it oscillated between 98 and 101 for a week. During the next three days the maximum daily temperature was around 100. After that time it dropped to normal, and remained around normal until the patient was discharged.

There was an extremely free flow of bile per catheter in the cystic duct. After the first few days, the patient was greatly relieved, and after four or five days was able to take liquids and semi-solids with relish.

The patient was discharged April 26, 1936, twenty-three days after admission to the hospital, and sixteen days after the operation. He was able to be up and about the room, and expressed himself as feeling better than he had felt for several years.

In this case we believe that it is logical to arrive at two distinct conclusions: (1) The primary pathology was in the bile-tract area, the pancreatitis being secondary to it. (2) The most important part of the operative procedure was the drainage of the pancreas by way of the common duct, the remains of the cystic duct and the catheter that was placed into it.

It is possible that satisfactory immediate results might have been obtained by doing a cholecystostomy, but, in view of the very distinct and gross evidences of gallbladder disease, it was decided that it would be best to remove the gallbladder because if it had not been removed it would have been a potential source of future difficulties.

We believe that, in the case of this patient, there was a partial obstruction of the common duct due to an extensive cholangitis.

L. L. and L. D. L.

## PLASTIC SURGERY

Edited by GEORGE H. KIMBALL, M.D., F.A.C.S.
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**Reconstructions About the Nasal Tip. Claire L. Straith, M.D., F.A.C.S., Detroit, Michigan. S. G. & O., Vol. 62, No. 1, January, 1936.**

The author states that procedures for correction of nasal deformities of all grades of severity have been greatly improved within recent times. As a result of this improvement, the patient requiring rhinoplasty, whatever the degree, can now be assured of a cosmetic or functional result sufficiently satisfactory to enable him to resume his rightful place in society.

The purpose of the article is to demonstrate the feasibility of successful rhinoplastic reconstructions without recourse to extrafacial sources for skin grafting, avoiding at the same time unsightly secondary facial blemishes. A new technique for the correction of nasal depressions which accomplishes its purpose without necessity of external incisions or from cartilage transplants is also presented. The Indian, or forehead flap, method, while an excellent means in women for covering major defects with skin of suitable color and texture, has definite disadvantages in the male. This method in men practically resolves itself into a matter of exchanging an ugly nasal defect for a less unsightly though nevertheless visible forehead scar. The Italian, or arm flap, method is mentioned only to be condemned. The skin provided has a color and texture which differs markedly from the facial integument. Large amounts of subcutaneous fat interferes with moulding and in healing as well.

To be acceptable, a surgical reconstruction of the nose must have a covering of smooth skin of normal color, a contour which harmonizes with the face, a functional epithelial lining and an adequate air way. Infection or malignant processes must be eradicated before repair is commenced; distorted tissues must be freed and replaced.

For reconstructions about the nasal tip the following methods are suggested:

### SKIN AND SOFT TISSUE DEFECTS

1. Small skin defects are readily covered by Wolfe grafts obtained from the upper eye lid or posterior aspect of the ear. This method is particularly useful when repair of the surface skin defects is attempted shortly after injury.

2. Defects of the ala nasi may be corrected by delayed pedicle flaps rolled down from the side of the nose, the normally rounded alar border being formed by the rolled edge. The resulting defect at the side of the nose is then covered by a Wolfe graft.

3. Skin and soft tissue losses at the nasal tip in women, especially when extensive, are best treated by forehead flap transfers, either by pedicles nourished by the temporal artery, or by the classical Indian method. In men the nasal tip is restored

by a tube pedicle from behind the ear by way of the sternal notch. This method has several advantages: (1) forehead scars are avoided, (2) the skin matches the nasal integument and is practically hairless, (3) the skin is thin and easily molded to shape, (4) the resulting neck scar is inconspicuous.

SUPPORTING STRUCTURE DEFECTS

Cartilage rather than prosthetic inserts of foreign substances should be used in reconstruction of the cartilaginous portion of the nasal bridge. Cartilage is not absorbed and has the advantage of being readily shaped and easily grafted.

1. Depressions of the cartilaginous bridge associated with prominence of the nasal tip can be filled in by one or more pieces of ear cartilage inserted through a mid-columellar incision.

2. Broadly depressed cartilaginous bridges associated with overhanging flattened nasal tips can be treated by several methods:

A. Hinged or mortise-jointed rib cartilage transplants may be inserted so that the vertical support rests upon the maxilla; the horizontal arm is shaped to correct the defects in the nasal bridge.

B. The introduction of rib cartilage transplants for depressions of the cartilaginous bridge can be avoided by the use of the author's recent extension of the principle of Kazanjian operation, (eversion of the lateral wings of the alar cartilage and suturing them back to back to support the nasal tip). The author augments this procedure by including also the upper lateral cartilages which are first cut, according to the depth of the bridge depression and then everted. They are next stitched back to back with two sutures of chromic catgut. Horsehair mattress sutures passed through the everted cartilage flaps and tied over rolls of gauze help to maintain their upright position. The horsehair sutures are removed in one week.

This method combined with that of the Kazanjian operation (which merely elevates and narrows the nasal tip) has produced excellent corrections of overhanging flattened nasal tips associated with broadly depressed cartilaginous bridges. It has the advantage of eliminating the necessity of rib resections as a source of cartilage, a disabling procedure which is not devoid of danger. It also forms a more flexible and natural nasal bridge than is obtained from the use of rib cartilage transplants. Moreover, the advantage of permitting the surgeon to shorten the nose and to elevate the bridge in a single stage operation without the necessity of external incisions, represents a distinct advantage over the more complicated and hazardous procedures hitherto described. The absence of a nasal septum strong enough to support the everted cartilages is an important contra-indication to this procedure.

COMMENT: The methods described undoubtedly are an advantage in reconstructions about the nose. They are not altogether new. Small skin defects about the nose may be covered with a skin graft immediately after injury using skin from the upper eyelid, back of the ear, or side of the neck.

This method insures a more nearly perfect skin texture of the graft.

It is well to avoid the disfiguring forehead scar in reconstructions of the nasal tip especially in men. However, in building a new nose or a large portion of a nose it is best to use the forehead material.

Most everyone agrees that cartilage is to be preferred in reconstructing the nasal tip. Ivory, paraffin and inert substances are to be condemned.

**Tannic Acid and Silver Nitrate in Burns. Adalbert G. Bettman, M.D., F.A.C.S., Portland, Oregon. S. G. & O., February 15, 1936.**

Dr. Bettman outlines the treatment for burns as follows:

1. The patient is given a narcotic which is repeated as often as may be necessary for comfort.

2. Fluids must be forced.

3. Grease or oil in any form should not have been used, but if such an application unfortunately has been made, it must be removed with ether or benzine before treatment is applied.

4. All blebs are to be opened and all loose skin and other burned tissue are to be removed.

A thorough application of five per cent tannic acid solution is made by means of cotton swabs. Following this, ten per cent silver nitrate solution is applied in the same manner. When this second solution is put on, a protective coagulum forms at once. Thus, a task that formerly required up to twenty-four hours is accomplished in a few minutes. The patient is placed in a tent heated by electric light bulbs and the areas treated are dried and kept dry. In a few days the coagulum begins to loosen and is removed as early as possible, a scalpel being used if necessary. Unhealed areas are treated by application of Oxyquinoline sulphate scarlet R gauze, the formula of which is:

| | | |
|---|---|---|
| R: Oxyquinoline sulphate | grains | 10 |
| Chlorbutanol (chloretone) | grains | 40 |
| Scarlet R ointment, 5% | ounces | 4 |
| Liquid petrolatum | drams | 4 |

The advantages claimed for this method are, a rapid tanning, avoidance of frequent dressings, keeping the patient dry and the speeding up of epithelial growth.

The author claims that under this treatment, skin grafts and other secondary corrective measures are very greatly reduced. The author claims that under the old method of using tannic acid in burns, there occurred an absorption of substances which affect the kidneys, liver, spleen, adrenals and other internal organs. He states that such effects have not been noted in patients who have been treated with tannic acid and silver nitrate, in the absence of grease.

When moisture is applied to a wound over a period of hours infection develops and tissues become edematous with some further breaking down. The new method avoids this complication.

Instead of the usual blood concentration, the hemoglobin and red cell count returns early to normal. The white blood cell count is low, the staff cells being relatively few throughout the entire healing period.

The author has used the tannic acid and silver nitrate method for two years and he lists the following advantages secured by the new method:

1. Saving lives that would be lost through slower method of tanning.

2. Immediate stopping of loss of body fluids, thereby preventing consequent concentration of blood.

3. Immediate prevention or very definite minimizing of shock.

4. Immediate prevention of absorption of toxic products.

5. Prevention of infection by the short period of application of moisture and the early drying of tanned tissues.

6. Saving of the kidneys and other organs from the effects of fluid concentration and the absorption of toxins and infection.

7. The greater comfort of the patient.

8. The fact that the patient is safely carried past the first twenty-four hours, the most critical period following a serious burn.

9. The fact that the patient avoids the second critical period, that of infection, and late absorption of toxic products.

10. The simplification of nursing problem, especially in the first twenty-four hours.

11. The prevention of further breaking down of tissues, resulting from long application of wet dressings.

12. The prevention of chilling, resulting from the long application of cold, wet dressings.

13. The formation of a thin, flexible coagulum.

14. The speedy healing of the burned areas with shortened period of hospitalization.

15. The prevention or minimizing of heavy contracting scars by early rapid healing in the absence of infection.

16. The lessening of the amount of skin grafting and secondary corrective surgery.

## ORTHOPAEDIC SURGERY

Edited by Earl D. McBride, M.D.
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**Tuberculosis of the Shafts of Long Bones: Clinical and Roentgenological Study with a Report of Eight Cases. C. K. Petter and J. B. Medelman. Amer. Rev. Tuberc. XXXII, 285, 1935.**

Of the 4,896 patients admitted to Glen Lake Sanatorium, 275 had bone and joint tuberculosis, with or without pulmonary manifestations. Of this latter number, eight patients had tuberculosis of the shafts of the long bones. This type of lesion, therefore, is extremely rare. The authors believe that tuberculosis of the shafts of long bones is primarily destructive in the cancellous portion, becoming proliferative when the periosteum is approached. In the differential diagnosis of lesions in the shafts of long bones, tuberculosis, syphilis, and pyogenic osteomyelitis should always be considered.

**The History of Plaster-of-Paris in the Treatment of Fractures. J. J. Monro. Brit. J. Surg., XXIII, 257, 1935.**

At least four thousand years ago, starch was being used in linen to give a stiffening effect for splinting.

Three phases in the use of plaster are recognized. Cheselden (1688-1752) and Larrey (about 1798) concentrated their attention to the bone. Later Seutin (1834) and Lucas-Championniere gave prior consideration to the soft parts. Movements of joints and massage are emphasized and light, portable starch apparatus was used. It remained for Krause and Bohler to point out that efficient splinting of fracture would allow the limb to be kept constantly in use.

Matthysen in 1852 first described the use of powdered plaster-of-Paris in treating fractures. When the time came for the removal of the bandage, this was done by wetting the plaster and unwinding the bandage.

**Spondylitis Anylopoeietica (Spondylitis Ossificans Ligamentosa). F. Campbell Golding. Brit. J. Surg., XXIII, 484, Jan., 1936.**

Rigidity of the spine from this condition may appear in any part, but the Marie-Strumpell type in the lumbar spine and the Bechterew type in the upper dorsal spine are the forms about which most has been written.

Infection is commonly believed to be the cause of the condition, although proof is lacking. Occupation, trauma, or strain do not appear to play a part. Males are most commonly affected, and the average age is the early thirties. The writer believes that the lesions occur first in the sacro-illiac joints; the smaller joints of the spine are then involved, with secondary involvement of the vertebral bodies and ligaments There is extensive connective-tissue formation with final ossification of the articular surfaces.

The writer, a roentgenologist, has collected the roentgenograms of ninety-one patients suffering from spondylitis ankylopoeietica. In every case, the sacro-iliac joints were involved, while in thirty-three cases, the sacro-iliac joints were involved, but the spine was unaffected. The symptoms in these latter patients were the same as the early symptoms in the more advanced group.

All patients gave a history of muscular and joint pains long before the onset of spinal rigidity. The author believes that the condition should be recognized earlier, that the sacro-iliac joints are the earliest clues to the diagnosis and to the degree of severity of the disease.

In some patients, the lesions burn themselves out and cease to give symptoms. Other patients get periods of relief from no treatment. Very little is said about the active treatment, but this omission is excusable because the article has been based on the roentgenographic study of the condition.

## INTERNAL MEDICINE

Edited by C. E. Bradley, M.D., Medical Arts Building, Tulsa; Hugh Jeter, M.D., 1200 North Walker, Oklahoma City

By HUGH JETER, M.D.

**Clinical Observations with Insulin Protamine Compound. Randall G. Sprague, M.D., Fellow in Medicine, Mayo Foundation; Benjamin B. Blum, M.D., Fellow in Medicine, Mayo Foundation; A. E. Osterberg, Ph.D., Edwin J. Kepler, M.D., and Russell M. Wilder, M.D., Rochester, Minn.**

This is the third report which has appeared in Journal, A. M. A., on insulin protamine compound, a new form of insulin which has been distributed to a good many doctors for clinical trial and which will soon be on the market. In this article it is called "insulin P" and the regular insulin such as we have been using "insulin R."

The results of their investigations are summarized as follows: The immediate effect of insulin-P (insulin protamine compound) is much less than that of insulin-R (regular insulin). When the former is used alone, and given as a single dose before breakfast, the meals of the first few days provoke glycosuria, but when the dose is properly adjusted the level of the blood sugar on successive mornings decreases progressively and the elevating effect of meals diminishes until, by the end of from four to six days, a normal level of blood sugar may be attained even in cases of severest diabetes.

Supplementing insulin-P with small doses of insulin-R will shorten the period of obtaining control. Insulin-R should not be mixed in the same syringe or injected into the same site with insulin-P. It has not been necessary to continue the supplementary use of insulin-R after the first few

days in the milder cases, but probably in many cases such supplementary use of insulin-R will be desirable, especially in emergencies. Until more experience has been obtained it would appear that insulin-R will be the insulin of choice when quick action is desirable, as in the treatment of acidosis.

Although insulin-P, in many cases, makes possible effective management of diabetes with only one administration of insulin a day, and with less insistence on rigid control of the diet, its careless use or disregard of the diet is attended with danger.

COMMENT: Evidently an extraordinary achievement has been reached in preparing an improved form of insulin. However, this means that very soon we will be confronted with the problem of evaluating new products or drugs which will be introduced to many physicians by detail men and it appears that there will be some competition and that some of the new insulin will not be prepared as has the product represented in this paper. I believe, therefore, that it is fortunate that we have reports such as this one to rely upon.

In a recent report at the meeting of the American College of Physicians, Dr. Joslin cited several cases which were handled by the protamine insulin more satisfactorily than by the regular insulin.

### Glycosuria and Hyperglycemia in Coronary Thrombosis

With the purpose of determining the frequency of disturbed carbohydrate tolerance during the acute phase of coronary closure and to prove or disprove the existence of a latent diabetes in these cases Adolph P. Raab and Meyer A. Rabinowitz, Brooklyn (Journal A. M. A., May 16, 1936), studied twenty-one patients, seventy-one per cent of whom gave evidence of abnormal sugar tolerance; but when only those twelve cases which were examined within two weeks of their coronary closure were considered, an abnormal sugar response was found in every instance. Since known cases of diabetes were excluded from this series it becomes evident that soon after the occurrence of a coronary closure the finding of an abnormal sugar tolerance curve is the rule. Of the nine cases in which a significant interval existed between the time of coronary closure and the dextrose tolerance test, only three gave an abnormal response. That sixty-seven per cent normal curves were found in this group in which an interval was permitted to elapse, while universally abnormal curves existed in the acute group, would argue against the existence of a latent diabetes as the predominant factor and speak for a temporary mechanism. The authors believe that a disturbance involving the vegetative nervous centers of the brain, as has also been suggested by Hausner and Hoff, is the important factor. The transient nature of the disturbed carbohydrate tolerance and its occurrence in cases in which neither pain nor shock was prominent would be consistent with this explanation. The appearance of glycosuria in numerous types of brain disorders, such as concussion, hemorrhage and tumor, would lend support to this belief. Insulin has been shown to be a drug that must be used with the greatest care in coronary closure. It is therefore most important to realize the existence of an underlying altered carbohydrate metabolism in these cases and to differentiate carefully coronary thrombosis with transient hyperglycemia and glycosuria from coronary closure and true diabetic acidosis, and from diabetic acidosis with electrocardiographic changes. As Scherf has pointed out, the question of a true diabetes mellitus cannot always be settled with certainty at the time of the attack of coronary thrombosis. The authors' study reemphasizes the importance of doing blood sugar estimations when the urine is negative for sugar. Root and Graybiel found that the prognosis of angina pectoris is worse in the diabetic patient and that those diabetic patients who died in the first year of their angina had received inadequate treatment for their diabetes.

### The Waterhouse-Friderichsen Syndrome

E. E. Aegerter, New York (Journal A. M. A., May 16, 1936), adds a case of his own and one case from the records of the Willard Parker Hospital which in history, symptomatology and pathology give the typical characteristics of the Waterhouse-Friderichsen syndrome. The syndrome was first accurately described in the literature of 1901. Since that time it has been recognized in England and Germany as a disease entity. The symptomatology includes sudden onset, malaise, restlessness, and often gastro-intestinal symptoms. These are followed shortly by lethargy, which rapidly deepens into coma. High fever, weak, rapid pulse, intense cyanosis and purpuric hemorrhages into the skin are characteristic. The disease is usually fatal in from sixteen to twenty-four hours. Massive, bilateral adrenal hemorrhage is the most common postmortem observation. The etiology is probably a fulminating meningococcemia. Suggested therapy includes adrenal cortex extract, epinephrine, sodium chloride, fluids, antimeningococcus serum, dextrose and transfusions.

### The Summer-Time Use of Mead's Oleum Percomorphum

During the hot weather, when fat tolerance is lowest, many physicians have found it a successful practice to transfer cod liver oil patients to Mead's Oleum Percomorphum.

Due to its negligible oil content and its small dosage, this product does not upset the digestion, so that even the most squeamish patient can "stomach" it without protest.

There are at least two facts that strongly indicate the reasonableness of the above suggestion: (1) In prematures, to whom cod liver oil cannot be given in sufficient dosage without serious digestive upset, Mead's Oleum Percomorphum is the antiricketic agent of choice. (2) In Floriad, Arizona and New Mexico, where an unusually high percentage of sunshine prevails at all seasons, Mead's Oleum Percomorphum continues increasingly in demand, as physicians realize that sunshine alone does not always prevent or cure rickets.

Mead Johnson & Company, Evansville, Indiana, invite you to send for samples of Mead's Oleum Percomorphum for clinical use during the summer months to replace cod liver oil.

### Summary of Regional Ileitis: Report of Case of Colonic Involvement and Suggestion of New Term

The case that A. J. Rosenblate, A. A. Goldsmith and A. A. Strauss, Chicago (Journal A. M. A., May 23, 1936), report shows a typical clinical and roentgenologic picture of regional ileitis, with added involvement up to but not including the distal portion of the transverse colon. The patient had been previously treated for four years for subacute bacterial endocarditis (despite negative blood cultures) and renal disease. The picture of regional ileitis is that of dull pain in the right lower quadrant, low grade intermittent fever, slight diarrhea, anorexia, anemia and rapid pulse. Roentgenologic studies of the ileum reveal characteristic manifestations. In cases in which the pathologic condition extends to the colon, the authors suggest the term "ileocolitis ulcerosa chronica." The treatment of choice is surgery.

### County-wide Use of Immune Globulin in Modification and Prevention of Measles

According to E. G. McGavran, Hillsdale, Mich. (Journal A. M. A. May 23, 1936), in the face of a predicted epidemic of measles of unusual proportion, the Hillsdale County Medical Society met the problem by distributing immune globulin, free of charge to the physicians. In return the physicians kept a careful record of the cases and turned them in to the secretary of the county medical society for analysis and conclusions. From three to five cc. of immune globulin was given intramuscularly or twelve cc. orally, when possible between the fourth and ninth day after exposure. Modification and no measles was considered as success. Only cases of definite exposure and definite history of susceptibility were included in the group in which measles did not develop. No patients less than six months of age are included in the series. Those receiving the usual intramuscular administration of the serum (seventy-seven cases in all) show ninety-five per cent success to five per cent failure. There were sixteen patients who received oral administration, twlve cc. of immune globulin in three four cc. doses in ice water before breakfast. Modified measles was present in twelve cases, no measles in one and in three there was no effect. After intramuscular administration, slight local reaction occurred in thirty-two, or roughly, one-third of the cases. Slight febrile reaction was observed in less than one-sixth of the cases. These reactions occurred in from twelve to twenty-four hours and did not last. No severe or general reaction was reported. The three chief variables are: amount of exposure, amount of immune globulin, and the time of administration. The last remains the most important single factor, but with intimate exposure in homes, an increased dosage or an earlier dosage is necessary to produce modification. In casual exposure it is wise to prolong the time before administration or lessen the amount of immune globulin to produce modification. A fourth factor may be considered as age. Older patients need more immune globulin and earlier administration to produce the same results.

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### Increased Metabolism of Obesity: Use and Abuse of Metabolic Stimulants

James J. Short and Harry J. Johnson, New York (Journal A. M. A., May 23, 1936), state that with increasing weight there is concomitantly an increasing total resting metabolism. That is, a person produces more heat when overweight than at normal weight. For instance, in the individual at normal weight under basal conditions heat is being produced at a certain rate per unit of surface area. When the same person increases his weight the surface area is also increased to accommodate this additional fat. If the heat production per unit of surface area is the same in the two instances, the total heat production in the obese state is obviously greater This relationship between weight and total metabolism has long been known but has been obscured by the emphasis that has been placed on basal metabolism to the exclusion of the total. Since in obesity there is already an increased total metabolism, the giving of metabolic stimulants in the form of thyroid preparations or drugs such as dinitrophenol is entirely illogical. At this stage such stimulants are not well tolerated. Thyroid extract caused toxic symptoms to become manifest very rapidly in a number of cases, although the explanation at the time was not well understood. As pointed out by Evans and Strang, many obese patients with high total heat production already manifest certain toxic symptoms characteristic of exophthalmic goiter. Additional stimulation would seem to be unwarranted and dangerous. A series of patients ranging from one to one hundred and thirty-five per cent overweight has been studied as to surface area, basal metabolism, total heat production, pulse rate, blood pressure and pulse pressure. The basal metabolism in obesity is usually within normal limits but tends to be in the lower ranges of normal for the slightly obese and in the higher ranges for the excessively obese. The increased metabolism favors rapid weight loss on dietary restriction. Thyroid preparations may be employed judiciously in later stages of treatment with submaintenance diets when it can be shown or assumed that the metabolic rates have been lowered. In all metabolic studies the heat production per unit of body surface (the so-called basal metabolism) and the total heat production of the organism under basal conditions should be sharply differentiated, since they represent two entirely different approaches to the problem of obesity. A study of the pulse rate has shown that those cases presenting a resting rate over eighty were definitely more overweight than those presenting lower rates, and that the total metabolism was likewise correspondingly higher.

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### The Physiologic Approach to Otolaryngology

Ralph A. Fenton, Portland, Ore. (Journal A. M. A., May 23, 1936), points out that the physiologic approach to otolaryngology was for many years a matter of measurement of motor or sensory functions. Of recent years, experimental observations have extended our knowledge of the cellular activities of normal and diseased ciliated membranes and have explained the growth, destruction and regeneration of these structures. The study of artificial sinus infections has disclosed the etiology of cyst and abscess formations, of osteomyelitis of the frontal sinus, of epidural abscess. In otology, mensuration of cochlear function has been carried into the eighth nerve, tone by tone, through electrical amplification. The ossicular muscles have been studied under the dissection microscope and their specific responses to sound stimuli accurately reported. From the vestibular system, six separate pathways for forced movements have been worked out, and the complex anatomy of the cerebellum has been unraveled through careful study of its developing tracts in the lowest vertebrates. The use of human immune serum instead of vaccines, and of transfusion instead of jugular ligation, has offered hope in recent otologic practice, in meningitis and in sinus thrombosis. Knowledge of the fundamentals of otolaryngologic physiology, interrelating various new discoveries and applying them to our field, will not teach us anything about surgical indications or surgical technic. But it may lead to a better understanding of the need for surgery and will be of great help in the application of modernized principles of immunity for the prophylaxis of disease of the upper respiratory tract. Otolaryngology in America will continue its progress, through insistence on higher standards of individual preparation, and greater interest by individuals thus prepared in the physiologic relationship of our special field to the various systems of the body as a whole. Team work with other members of hospital staffs often points the way to interesting bits of research. Each one, however far away from others in the specialty, should feel that he is a collaborator, through his reading and his papers before local medical groups, in the task of apprizing the general profession that we are not immediately bent on drastic surgery but that we are building, through physiologic and pathologic knowledge, a sound foundation for the medical and surgical otolaryngology of the future.

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NOTE—Corrections and additions to the above list will be cheerfully accepted.

# ROSTER

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## 1935

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| BUTTS, A. J. | Cherokee |
| CLARK, Z. J. | Cherokee |
| DUNNINGTON, W. G. | Cherokee |
| HARRIS, G. G. | Helena |
| HUSTON, H. E. | Cherokee |
| LANCASTER, L. T. | Cherokee |
| MERCER, J. W. | Cherokee |

### ATOKA

| | |
|---|---|
| CLARK, J. B. | Coalgate |
| FULTON, J. S. | Atoka |
| GARDNER, C. C. | Ponca City |

### BECKHAM

| | |
|---|---|
| BAKER, L. V. | Elk City |
| BRIGHTWELL, GAINES | Leedey |
| DENBY, J. M. | Carter |
| DeVANNEY, P. J. | Sayre |
| JONES, C. F. | Erick |
| KILPATRICK, E. S. | Elk City |
| McCARTY, D. E. | Texola |
| McCREERY, R. C. | Erick |
| McGRATH, T. J. | Sayre |
| SPEED, Sr., H. K. | Sayre |
| SPEED, Jr., H. K. | Sayre |
| STAGNER, G. H. | Erick |
| TISDAL, V. C. | Elk City |
| TISDAL, W. C. | Elk City |

### BLAINE

| | |
|---|---|
| BARNETT, J. S. | Hitchcock |
| BROWNING, J. W. | Geary |
| CORDONNIER, B. J. | Okeene |
| COX, A. K. | Watonga |
| HART, E. E. | Canton |
| HILL, T. A. | Watonga |
| HOLCOMBE, GEO. M. | Okeene |
| MURDOCK, L. H. | Okeene |
| PADBERG, A. F. | Canton |

### BRYAN

| | |
|---|---|
| ARMSTRONG, DAVID | Durant |
| BAKER, ALFRED | Durant |
| BOLLINGER, E. W. | Achille |
| CAIN, P. L. | Albany |
| COCHRAN, ROY L. | Caddo |
| COKER, B. B. | Durant |
| COLWICK, J. T. | Durant |
| COLWICK, O. J. | Durant |
| DRAKE, T. L. | Durant |
| FUSTON, H. B. | Bokchito |
| HAYNIE, JOHN A. | Durant |
| HOUSER, W. A. | Durant |
| McCALIB, D. C. | Utica |
| McCARLEY, W. H. | Colbert |
| MOORE, B. H. | Perrine Bldg., Oklahoma City |
| MOORE, CHAS. F. | Durant |
| PRICE, CHAS. C. | Durant |
| RUSHING, G. M. | Durant |
| SAWYER, R. E. | Durant |
| SHULER, JAS. L. | Durant |
| TONY, S. M. | Bennington |
| WANN, C. E. | Albany |
| WELLS, A. J. | Calera |
| WHARTON, J. T. | Durant |

### CADDO

| | |
|---|---|
| ANDERSON, P. H. | Anadarko |
| BLAIR, SAMUEL | Apache |
| BUTLER, I. S. | Carnegie |
| CAMPBELL, GEO. C. | Anadarko |
| COOK, ODIS A. | Apache |
| HASLAM, G. E. | Anadarko |
| HAWKINS, E. W. | Binger |
| HAWN, W. T. | Apache |
| HENKE, J. J. | Hydro |
| HENRY, J. WORRELL | Anadarko |
| HUME, CHAS. R. | Anadarko |
| INMAN, E. L. | Apache |
| JOHNSTON, R. E. | Bridgeport |
| KELLEAM, E. A. | Sells, Arizona |
| KERLEY, W. W. | Anadarko |
| McCLURE, P. L. | Fort Cobb |
| McMILLAN, C. B. | Gracemont |
| MILES, J. B. | Anadarko |
| PUTNAM, W. B. | Carnegie |
| RECTOR, R. D. | Anadarko |
| ROGERS, F. W. | Carnegie |
| TAYLOR, A. H. | Anadarko |
| WILLIAMS, R. W. | Anadarko |
| WILLIAMS, S. E. | Hydro |

### CANADIAN

| | |
|---|---|
| ADERHOLD, THOS. M. | El Reno |
| BROWN, HADLEY C. | El Reno |
| CATTO, W. B. | El Reno |
| CLARK, FRED H. | El Reno |
| CRADEN, PAUL J. | El Reno |
| DEVER, HARVEY A. | El Reno |
| DEVER, HARVEY K. | El Reno |
| FUNK, G. D. | El Reno |
| GOODMAN, GEO. LEROY | Yukon |
| HEROD, PHILIP F. | El Reno |
| JOHNSON, ALPHA L. | El Reno |

*Deceased

KUCHAR, V. ..... Yukon
LAWTON, W. P. ..... El Reno
MILLER, W. R. ..... Calumet
MUZZY, WM. J. ..... El Reno
MYERS, PIRL B. ..... El Reno
PHELPS, JOS. T. ..... El Reno
PHELPS, MALCOM E. ..... El Reno
RILEY, JAS. T. ..... El Reno
RICHARDSON, D. P. ..... Union City
STOUGH, Sr., D. F. ..... Geary
TOMKINS, JOHN E. ..... Yukon
WOLF, L. G. ..... Okarche

### CARTER

BAKER, E. R. ..... Healdton
BOADWAY, F. W. ..... Adrmore
CANTRELL, D. E. ..... Wilson
CANTRELL, E. J. ..... Wilson
COX, J. L. ..... Ardmore
EASTERWOOD, A. Y. ..... Ardmore
GILLISPIE, L. D. ..... Springer
HARDY, WALTER ..... Ardmore
HATHAWAY, W. G. ..... Lone Grove
HIGGINS, H. A. ..... Ardmore
JACKSON, T. J. ..... Ardmore
JOHNSON, C. A. ..... Wilson
JOHNSON, G. E. ..... Ardmore
JOHNSON, WALTER ..... Adrmore
LOONEY, MacDONALD ..... Marietta
MOTE, W. R. ..... Ardmore
McNEES, J. C. ..... Ardmore
PARRISH, R. M. ..... Ardmore
PATTON, DOYLE C. ..... Tishomingo
PERRY, FRED T. ..... Healdton
POLLOCK, J. R. ..... Ardmore
SULLIVAN, R. C. ..... Ardmore
VEAZY, J. C. ..... Ardmore
VEAZY, J. H. ..... Ardmore
WATERS, C. B. ..... Marietta
WOODS, L. B. ..... Ardmore

### CHEROKEE

ALLISON, J. S. ..... Tahlequah
BAINES, SWATRZ ..... Tahlequah
MEDEARIS, P. H. ..... Tahlequah

### CHOCTAW

GEE, R. L. ..... C. C. C. Camp, Thermopolis, Wyo.
HALE, C. H. ..... Boswell
HARRIS, G. E. ..... Hugo
JOHN, W. N. ..... Hugo
JOHNSON, E. A. ..... Hugo
*MILLER, J. S. ..... Hugo
SHULL, R. J. ..... Hugo

### CLEVELAND

ATKINS, W. H. ..... Norman
BOBO, C. S. ..... Norman
BOND, I. T. ..... Norman
BRAKE, C. A. ..... Norman
CLIFTON, G. M. ..... Norman
COOLEY, B. H. ..... Norman
GABLE, J. J. ..... Norman
GRIFFIN, D. W. ..... Norman
HADDOCK, Jr., J. L. ..... Norman
HOOD, J. O. ..... Norman
HOWELL, O. E. ..... Norman
KNISELY, H. B. ..... Norman
LAMBERT, J. B. ..... Lexington
LOWTHER, R. D. ..... Norman
MAYFIELD, W. T. ..... Norman

*Deceased

MERRITT, I. S. ..... Norman
RAYBURN, C. R. ..... Norman
REICHERT, R. J. ..... Moore
REIGER, J. A. ..... Norman
SCHMIDT, ELEANORA ..... Norman
STEEN, CARL ..... Norman
STEPHENS, E. F. ..... Norman
THACKER, R. E. ..... Lexington
WICKHAM, M. M. ..... Norman
WILEY, G. A. ..... Norman
WILLARD, D. G. ..... Norman
TURLEY, L. A. ..... Norman

### COMANCHE

ANGUS, DONALD ..... Lawton
ANGUS, H. A. ..... Lawton
ANTONY, JOS. T. ..... Lawton
BARBER, GEO. S. ..... Lawton
BROSHEARS, JACKSON ..... Lawton
DUNLAP, E. B. ..... Lawton
FERGUSON, L. W. ..... Lawton
GOOCH, L. T. ..... Lawton
HAMMOND, F. W. ..... Lawton
HATHAWAY, E. P. ..... Lawton
HUGHES, C. P. ..... Lawton
KERR, GEO. E. ..... Chattanooga
KNEE, L. C. ..... Lawton
LUTNER, THOS. R. ..... Lawton
MALCOLM, J. W. ..... Lawton
MARTIN, CHAS. M. ..... Elgin
MASON, W. J. ..... Lawton
MITCHELL, E. BRENT ..... Lawton
PARSONS, O. L. ..... Lawton

### COTTON

BAKER, G. W. ..... Walters
HARNED, W. B. ..... Walters
HOLSTEAD, A. B. ..... Temple
HOUSE, C. F. ..... Walters
JANES, G. M. ..... Walters
JONES, M. A. ..... Walters
McKINNEY, H. ..... Temple
SCISM, MOLLIE F. ..... Walters

### CRAIG

ADAMS, F. M. ..... Vinita
BAGBY, LOUIS ..... Vinita
BAUM, E. ELDON ..... Vinita
BRADSHAW, J. O. ..... Welch
DARROUGH, J. B. ..... Vinita
ELAM, B. L. ..... Centralia
GASTINEAU, F. T. ..... Vinita
HAYES, P. L. ..... Vinita
HERRON, A. W. ..... Vinita
LEHMER, ELIZABETH ..... Vinita
MARKS, W. R. ..... Vinita
MITCHELL, R. L. ..... Veterans Hospital, Muskogee
NEER C. S. ..... Vinita
STOUGH, D. B. ..... Vinita
WALKER, C. F. ..... Grove

### CREEK

BISBEE, W. G. ..... Bristow
CARGILL, J. E. ..... Shamrock
COFFIELD, A. W. ..... Drumright
COPPEDGE, O. C. ..... Bristow
COPPEDGE, O. S. ..... Depew
COWART, O. H. ..... Bristow
CROSTON, GEO. C. ..... Sapulpa
CURRY, J. F. ..... Sapulpa
DRIVER, C. M. ..... Mounds
HAAS, H. R. ..... Sapulpa
HOLLIS, J. E. ..... Bristow

HUMPHREYS, D. W. ..... Oilton
JONES, ELLIS ..... Sapulpa
KING, E. W. ..... Bristow
LAMPTON, J. B. ..... Sapulpa
LEWIS, P. K. ..... Sapulpa
LONGMIRE, W. P. ..... Sapulpa
McCALLUM, C. L. ..... Sapulpa
McDONALD, C. B. ..... Mannford
MOTE, PAUL ..... Sapulpa
NEAL, WM. J. ..... Drumright
REESE, C. B. ..... Sapulpa
REYNOLDS, E. W. ..... Bristow
REYNOLDS, S. W. ..... Drumright
SCHRADER, CHAS. ..... Bristow
SCHWAB, B. C. ..... Sapulpa
SISLER, FRANK H. ..... Bristow
STARR, O. W. ..... Drumright
TURNER, F. W. ..... Sapulpa
WELLS, JOHN M. ..... Bristow
WHARTON, J. L. ..... Depew
WHITE, EDW. E. ..... Oilton
WILLIAMS, J. CLAY ..... Bristow

## CUSTER

ALEXANDER, C. J. ..... Clinton
ALLEN, FRANK W. ..... Leedey
BAGLEY, JAMES A. ..... Clinton
BASINGER, W. I. ..... Butler
BOYD, T. A. ..... Weatherford
DOLER, C. ..... Clinton
EDWARDS, E. C. ..... Clinton
FRIZZELL, J. T. ..... Cunton
GOSSOM, K. D. ..... Custer City
HICKMAN, R. L., 162 Hatton Ave., Lebanon, Tenn
HINSHAW, J. R. ..... Butler
LAMB, ELLIS ..... Clinton
LAMB, LEALON ..... Clinton
LOYD, E. M. ..... Talogo
McBURNEY, C. H. ..... Clinton
PAULSON, A. W. ..... Clinton
REDWINE, H. P. ..... Snyder, Texas
ROGERS, McLAIN ..... Clinton
RUHL, N. E. ..... Weatherford
RYAN, W. A. ..... Thomas
VIEREGG, FRANK R. ..... Clinton
WAIT, W. C. ..... Clinton
WILLIAMS, GORDON D. ..... Weatherford

## GARFIELD

AITKEN, W. A. ..... Enid
BAGBY, E. L. ..... Enid
BAKER, R. C. ..... Enid
CHAMPLIN, PAUL B. ..... Enid
COTTON, LEE W. ..... Enid
DUFFEY, FRANCIS M. ..... Enid
GILL, W. W. ..... Enid
GREGG, O. R. ..... Enid
FEILD, JULIAN ..... Enid
FRANCISCO, GLENN ..... Enid
FRANCISCO, J. W. ..... Enid
HARRIS, D. S. ..... Drummond
HAMBLE, V. R. ..... Enid
HARTMAN, G. W. ..... Sharon, Pa.
HINSON, BRUCE R. ..... Enid
HINSON, T. B. ..... Enid
HOPKINS, P. W. ..... Enid
HUDSON, F. A. ..... Enid
HUDSON, H. H. ..... Enid
JACOBS, R. G. ..... Enid
KENDALL, W. L. ..... Enid
KIEBLER, W. G. ..... Enid
LAMBERTON, W. E. ..... Enid
LINDSTROM, W. C. ..... Garber
MATTHEWS, G. F. ..... Tahlequah
MAYBERRY, S. N. ..... Enid
McEVOY, S. H. ..... Enid
McINNIS, A. L. ..... Enid
NEILSON, W. P. ..... Enid
NEWELL, W. B. ..... Enid
PIPER, A. S. ..... Enid
RHODES, W. H. ..... Enid
ROBERTS, C. J ..... Enid
ROBERTS, D. D. ..... Enid
SHANNON, H. R. ..... Enid
SHEETS, MARION E. ..... Enid
SWANK, J. R. ..... Enid
VANDEVER, H. F. ..... Enid
WALKER, JOHN R. ..... Enid
WATSON, J. M. ..... Enid
WILKINS, A. E. ..... Covington
WOLFE, E. J. ..... Waukomis

## GARVIN

ALEXANDER, ROBT. M. ..... Paoli
BURNS, S. L. ..... Stratford
CALLAWAY, JOHN R. ..... Pauls Valley
GREENING, W. P. ..... Pauls Valley
GROSS, J. T. ..... Lindsay
JOHNSON, G. L. ..... Pauls Valley
LINDSEY, RAY H. ..... Pauls Valley
MONROE, HUGH H. ..... Lindsay
PRATT, CHAS. M. ..... Lindsay
ROBBERSON, M. E. ..... Wynnewood
SHI, A. H. ..... Stratford
SULLIVAN, C. L. ..... Elmore City
TAYLOR, E. F. ..... Maysville
WILSON, H. P. ..... Wynnewood
WINTER, J. D. ..... Wynnewood

## GRADY

AMBRISTER, J. C. ..... Chickasha
BARRY, J. R. ..... Alex
BAZE, W. J. ..... Chickasha
BONNELL, W. L. ..... Chickasha
BOON, U. C. ..... Chickasha
COOK, W. H. ..... Chickasha
COX, C. P. ..... Ninnekah
DOWNEY D. S. ..... Chickasha
EMANUEL, L. E. ..... Chickasha
EMANUEL, ROY ..... Chickasha
GERARD, G. R. ..... Chickasha
HAMPTON, P. J. ..... Rush Springs
HENNING, A. E. ..... Tuttle
LEEDS, A. B. ..... Chickasha
LITTLE, AARON ..... Minco
LIVERMORE, W. H. ..... Chickasha
MARRS, S. O. ..... Chickasha
MASON, REBECCA H. ..... Chickasha
McCLURE, H. M. ..... Chickasha
MITCHELL, C. P. ..... Chickasha
NUNNERY, A. W. ..... Chickasha
PYLE, OSCAR S. ..... Chickasha
RENEGAR, J. F. ..... Tuttle
WOODS, L. E. ..... Chickasha

## GRANT

GRIMAND, W. R. ..... Medford
HARDY, I. V. ..... Medford
LAWSON, E. E. ..... Medford
LIVELY, S. A. ..... Wakita

## GREER

BORDER, G. F. ..... Mangum
CHAMBERS, M. E. ..... Vinson
CHERRY, G. P. ..... Mangum
DODSON, W. O. ..... Willow
HOLLIS, J. B. ..... Mangum
LANSDEN, J. B. ..... Granite

LOWE, J. T. ..... Mangum
MEREDITH, J. S. ..... Duke
NELSON, H. J. ..... Granite
OLIVER, W. D. ..... Mangum
PEARSON, LEB. E. ..... Mangum
POER, E. M. ..... Mangum

### HARMON

ALLGOOD, E. J. ..... Gould
HOLLIS, LYNN E. ..... Hollis
HOPKINS, SAMUEL W. ..... Hollis
HUSBAND, WM. G. ..... Hollis
JONES, JAMES E. ..... Hollis
LYNCH, RUSSELL H. ..... Hollis
RAY, W. T. ..... Gould
YEARGAN, WM. M. ..... Hollis

### HASKELL

CARSON, WM. S. ..... Keota
HILL, A. T. ..... Stigler
RUMLEY, JAMES C. ..... Stigler
THOMPSON, WM. A. ..... Stigler
TURNER, THOS. BOYD ..... Stigler
WILLIAMS, N. K. ..... McCurtain

### HUGHES

BENTLEY, J. A. ..... Allen
BUTTS, A. M. ..... Holdenville
BUTTS, IMOGENE ..... Holdenville
DAVENPORT, A. L. ..... Holdenville
DIGGS, G. W. ..... Wetumka
FLOYD, W. E. ..... Holdenville
FORD, R. B. ..... Holdenville
FORSYTHE, THOS. ..... Allen
FRYE, HARRY ..... Holdenville
HAMILTON, S. H. ..... Non
HEMPHILL, J. A. ..... Wetumka
HICKS, C. A. ..... Wetumka
HOWELL, H. A. ..... Holdenville
MARTIN, C. S. ..... Calvin
PRYOR, V. W. ..... Holdenville
STONER, R. W. ..... Wetumka
TAYLOR, W. L. ..... Holdenville

### JACKSON

ABERNETHY, EDW. A. ..... Altus
ALLGOOD, JOHN ..... Altus
BIRD, JESSE, ..... Eldorado
BROWN, R. F. ..... Altus
CROW, E. S. ..... Olustee
ENSEY, J. E. ..... Altus
FOX, RAYMOND H. ..... Altus
HIX, JOSEPH B. ..... Altus
MABRY, EARL W. ..... Altus
McCONNELL, L. H. ..... Altus
McFADIN, J. S. ..... Altus
REID, JOHN R. ..... Altus
RUDELL, W. P. ..... Altus
SPEARS, CLAUD G. ..... Altus
STARKEY, WAYNE ..... Altus
TAYLOR, ROBT. Z. ..... Blair

### JEFFERSON

ANDRESKOSKI, W. P. ..... Ryan
BROWNING, W. M. ..... Waurika
COLLINS, D. B. ..... Waurika
DERR, J. I. ..... Waurika
EDWARDS, F. M. ..... Ringling
HOLLINGSWORTH, J. T. ..... Waurika
MAUPIN, C. M. ..... Waurika
McPHERSON, J. M. ..... Terrell
MINGUS, F. M. ..... Loco
WADE, L. L. ..... Ryan
WATSON, J. W. ..... Ryan

### JOHNSTON

CLARK, GUY ..... Milburn

### KAY

ARMSTRONG, W. O. ..... Ponca City
ARRENDELL, C. W. ..... Ponca City
BEATTY, J. H. ..... Tonkawa
BECKER, L. H. ..... Blackwell
BERRY, G. L. ..... Blackwell
BROWNE, H. S. ..... Ponca City
CLIFT, M. C. ..... Blackwell
DARNELL, E. E. ..... Toppenish, Wash.
DENHAM, T. W. ..... Three Sands
GIBSON, R. B. ..... Ponca City
GORDON, D. M. ..... Ponca City
GOWEY, H. O. ..... Newkirk
*HAWKINS, J. C. ..... Blackwell
HOWE, J. H. ..... Ponca City
HUDSON, J. O. ..... Braman
KREGER, G. S. ..... Tonkawa
*LESLIE, W. M. ..... Blackwell
MALL, WERNER W. ..... Palm Springs, Calif.
MATTHEWS, DEWEY ..... Tonkawa
McELROY, THOMAS ..... Ponca City
MILLER, D. W. ..... Blackwell
MOORE, G. C. ..... Ponca City
MORGAN, L. S. ..... Ponca City
NEAL, L. G. ..... Ponca City
NIEMANN, G. H. ..... Ponca City
NORTHCUTT, C. E. ..... Ponca City
NUCKOLS, A. S. ..... Ponca City
OBERMILLER, R. G. ..... Ponca City
PETERS, M. L. ..... Blackwell
RISSER, A. S. ..... Blackwell
SANGER, W. W. ..... Ponca City
SPENCE, H. M. ..... Dolos, Texas
STALEY, P. A. ..... Palm Springs, Calif.
VANCE, L. C. ..... Ponca City
WAGGONER, E. E. ..... Tonkawa
*WAGNER, J. C. ..... Ponca City
WALKER, I. D. ..... Tonkawa
WHITE, M. S. ..... Blackwell
YEARY, G. H. ..... Newkirk

### KINGFISHER

CAVETT, E. R. ..... Loyal
DIXON, A. ..... Hennessey
HODGSON, C. M. ..... Kingfisher
GOSE, C. O. ..... Hennessey
LATTIMORE, F. C. ..... Kingfisher
PENDLETON, JOHN W. ..... Kingfisher
STURGEON, H. VIOLET ..... Hennessey
TOWNSEND, B. I. ..... Hennessey
VINCENT, I. H. ..... Dover

### KIOWA

ADAMS, J. L. ..... Hobart
ADAMS, RICHARD ..... Hobart
ANDERSON, H. R. ..... Mountain View
BONHAM, J. M. ..... Hobart
BRAUN, J. P. ..... Hobart
BRYCE, J. R. ..... Snyder
FINCH, J. WM. ..... Sentinel
HATHAWAY, A. H. ..... Mountain View
LAND, J. A. ..... Hobart
LLOYD, H. C. ..... Hobart
MARTIN, F. F. ..... Roosevelt
MILES, E. P. ..... Snyder
MOORE, J. H. ..... Hobart
PRESTON, C. R. ..... Mountain Park
RITTER, J. M. ..... Roosevelt

*Deceased

WALKER, F. E. ..... Lone Wolf
WATKINS, B. H. ..... Hobart

LATIMER

HAMILTON, E. B. ..... Wilburton
HARRIS, J. M. ..... Wilburton
HENRY, R. L. ..... Wilburton
RICH, R. L. ..... Red Oak

LE FLORE

BAKER, F. P. ..... Talihina
BEVILL, S. D. ..... Poteau
BOOTH, G. R. ..... LeFlore
COLLINS, E. L. ..... Panama
DEAN, S. C. ..... Howe
DUFF, W. M. ..... Braden
FAIR, E. N. ..... Heavener
GILLIAM, W. C. ..... Spiro
HARTSHORNE, G. E. ..... Picner
HEAD, W. M. ..... Talihina
HENRY, M. L. ..... Heavener
MINOR, R. M. ..... Williams
ROLLE, NEESON ..... Poteau
SHIPPEY, W. L. ..... Poteau
WOODSON, E. M. ..... Poteau
WRIGHT, R. L. ..... Poteau

LINCOLN

ADAMS, J. W. ..... Chandler
ANDERSON, W. D. ..... Claremore
BAILEY, C. H. ..... Stroud
BAIRD, W. D. ..... Prague
BERRY, THOS. M. ..... C.C.C. Camp, Bogato, Texas
BURLESON, NED ..... Prague
DAVIS, W. B. ..... Stroud
HOWELL, C. H. ..... Meeker
JENKINS, H. B. ..... Tryon
MARSHALL, A. M. ..... Chandler
MURRAY, LEVI, 406 Federal Bldg., Oklahoma City
NICHOLS, U. C. ..... Davenport
NORWOOD, F. H. ..... Prague
ROBERTSON, C. W. ..... Chandler
ROLLINS, J. S. ..... Prague
*SOSBEE, J. W. ..... Stroud

LOGAN

ALLEN, ROBT. ..... Guthrie
BARKER, C. B. ..... Guthrie
BARKER, E. O. ..... Guthrie
BARKER, PAULINE ..... Guthrie
CORNWELL, N. L. ..... Coyle
FIRST, F. R. ..... Crescent
GARDNER, P. B. ..... Marshall
GOODRICH, E. E. ..... Crescent
GRAY, DAN ..... Guthrie
HAHN, L. A. ..... Guthrie
HILL, C. B. ..... Guthrie
LARKIN, W. H. ..... Minco
LE HEW, J. L. ..... Guthrie
MILLER, WM. C. ..... Guthrie
PETTY, C. S. ..... Guthrie
RINGROSE, R. F. ..... Guthrie
RITZHAUPT, L. H. ..... Guthrie
SOUTER, J. E. ..... Guthrie
TRIGG, F. E. ..... Guthrie
WEST, A. A. ..... Guthrie

MAJOR

CAMPBELL, JOHN D. ..... Fairview
SCOTT, G. E. ..... Ringwood
SPECHT, ELSIE ..... Fairview

*Deceased

MARSHALL

HAYNIE, W. D. ..... Kingston
HOLLAND, J. L. ..... Madill
LOGAN, J. H. ..... Lebanon
ROBINSON, P. F. ..... Madill
YORK, J. F. ..... Madill

MAYES

ADAMS, SYLBA ..... Hayward, Wis.
BRYANT, W. C. ..... Choteau
HACKLER, H. W. ..... Pryor
HERRINGTON, V. D. ..... Pryor
HILLIS, J. E. ..... Pryor
HOLLINGSWORTH, J. E. ..... Strang
MORROW, B. L. ..... Salina
PUCKETT, CARL ..... 22 W. Sixth, Oklahoma City
RUTHERFORD, V. M. ..... Pryor
WHITAKER, W. J. ..... Pryor
WHITE, L. C. ..... Adair

McCLAIN

BARGER, G. S. ..... Purcell
COCHRANE, J. E. ..... Byars
DAWSON, O. O. ..... Wayne
KOLB, I. N. ..... Blanchard
McCURDY, W. C. ..... Purcell
ROYSTER, R. L. ..... Purcell

McCURTAIN

BARKER, N. L. ..... Broken Bow
CLARKSON, A. W. ..... Valliant
HALL, LYMAN S. ..... Broken Bow
MORELAND, J. T. ..... Idabel
MORELAND, W. A. ..... Idabel
SHERRILL, R. H. ..... Broken Bow
WILLIAMS, R. D. ..... Idabel

McINTOSH

JACOBS, L. I. ..... Hanna
LEE, N. P. ..... Checotah
LITTLE, D. E. ..... Eufaula
SMITH, F. L. ..... Eufaula
TOLLESON, W. A. ..... Eufaula
WOOD, JAS. L. ..... Eufaula

MURRAY

ANADOWN, P. V. ..... Sulphur
BAILEY, H. C. ..... Sulphur
BALL, ERNEST ..... Sulphur
BURKE, RICHARD M. ..... Sulphur
DUNHAM, H. C. ..... Sulphur
LUSTER, J. C. ..... Davis
POWELL, W. H ..... Sulphur
SADLER, F. E. ..... Sulphur
SHIVERS, E. E. ..... Davis
SLOVER, GEO. ..... Sulphur
SPROUSE, O. W. ..... Sulphur

MUSKOGEE

BALLANTINE, H. T. ..... Surety Bldg.
BERRY, W. D. ..... Barnes Bldg.
BLAKEMORE, J. L. ..... Barnes Bldg.
BALLANCE, R. A. ..... Webber Falls
BURNS, H. C. ..... Warner
BRUTON, L. D. ..... Commercial Natl. Bldg.
BRYAN, CECIL ..... Vian
CAMPBELL, J. F. ..... Barnes Bldg.
COACHMAN, E. H. ..... Manhattan Bldg.
DE GROOT, C. E. ..... Manhattan Bldg.
DONNELL, R. N. ..... Raymond Bldg.

DORWART, F. G. ..... Barnes Bldg.
EARNEST, A. N. ..... Barnes Bldg.
EVERLY, A. W. ..... Equity Bldg.
EWING, F. W. ..... Surety Bldg.
FITE, E. H. ..... Barnes Bldg.
FITE, W. P. ..... Barnes Bldg.
FRYER, S. J. ..... Surety Bldg.
FULLENWIDER, C. M. ..... Barnes Bldg.
GRAVES, J. R. ..... Boynton
HAMM, S. G. ..... Haskell
*HARROD, R. T. ..... Oktaha
HEITZMAN, C. W. ..... Barnes Bldg.
HOLCOMB, R. N. ..... Surety Bldg.
JOBLIN, W. R. ..... Porter
KAISER, GEO. L. ..... Checotah
KLASS, O. C. ..... Surety Bldg.
KUPKA, J. F. ..... Haskell
McALISTER, L. S. ..... Barnes Bldg.
MOBLEY, A. L., Vets. Facility, Albuquerque, N. M.
NEELY, S. D. ..... Commercial Natl. Bldg.
NEWHAUSER, MAYER ..... Vet. Hosp., Muskogee
NICHOLS, J. T. ..... Equity Bldg.
NOBLE, JOS. G. ..... 1400 E. Okmulgee
OLDHAM, Sr., I. B. ..... 426 North Sixth
OLDHAM, Jr., I. B. ..... 426 North Sixth
OSGOOD, W. W. ..... Equity Bldg.
RAFTER, J. G. ..... Manhattan Bldg.
REYNOLDS, JOHN ..... First Natl. Bldg.
RICE, C. V. ..... 1620 West Okmulgee
SANSING, CAMPBELL, Vet. Hospital, Muskogee
SCOTT, H. A. ..... Commercial Natl. Bldg.
THOMAS, L. M. ..... Webber Falls
THOMPSON, M. K. ..... Surety Bldg.
TILTON, W. B. ..... Vet. Hospital, Muskogee
WALLIS, G. F. ..... Fort Gibson
WARTERFIELD, F. E. ..... Commercial Natl. Bldg.
WHITE, C. E. ..... 562 North Sixth
WHITE, J. H. ..... Surety Bldg.
WILKIEMEYER, F. J. ..... Vet. Hospital, Muskogee
WOLFE, I. C. ..... 426 North Sixth
WOODBURN, J. T. ..... Surety Bldk.
WOODNICK, GEO. W. ..... Vet. Facility, Muskogee

## NOBLE

COOKE, C. H. ..... Perry
EVANS, A. M. ..... Perry
FRANCIS, J. W. ..... Perry
RENFROW, T. F. ..... Billings

## NOWATA

LANG, S. A. ..... Nowata
PRENTISS, H. M. ..... Nowata
ROBERTS, S. P. ..... Nowata
SCOTT, M. B. ..... Delaware
SUDDERETH, J. P. ..... Nowata

## OKFUSKEE

ADAMS, ALLEN C. ..... Elk City
BARTOW, A. C. ..... Okfuskee
BLOSS, C. M. ..... Okemah
BOMBARGER, C. C. ..... Paden
BRICE, M. O. ..... Okemah
COCHRAN, C. M. ..... Okemah
DOVELL, J. C. ..... Paden
JENKINS, W. P. ..... Okemah
KENNEDY, J. A. ..... Okemah
KEYES, ROBERT ..... La Mesa, Calif.
LUCAS, A. C. ..... Castle
MELTON, A. S. ..... Okemah
PEMBERTON, J. M. ..... 217½ West Commerce Oklahoma City
PRESTON, J. R. ..... Weleetka
SPICKARD, L. J. ..... Okemah

*Deceased

## OKLAHOMA

ADAMS, ROBT. H. ..... 501 Ramsey Tower
AKIN, R. H. ..... 400 West Tenth St.
ALFORD, J. M. ..... Medical Arts Bldg.
ALLEN, E. P. ..... 1200 North Walker
ALLEN, GEO. T. ..... Osler Bldg.
ANDREWS, LELIA E. ..... 1200 North Walker
ARRINGTON, C. T. ..... 805 North Walnut
BAILEY, F. M. ..... Minco
BAILEY, WILLIAM H. ..... 301 West Twelfth St.
BAIRD, W. D. ..... 2519½ South Robinson
BALYEAT, RAY M. ..... 1200 North Walker
BARKER, CHAS. E. ..... 505-13 Osler Bldg.
BARB, THOS. J. ..... 240 West Commerce
BATES, C. E. ..... Federal Bldg.
BATCHELOR, J. J. ..... Medical Arts Bldg.
BERRY, C. N. ..... Medical Arts Bldg.
BEYER, M. R. ..... 2006 North West 39th St.
BINKLEY, JAMES G. ..... Medical Arts Bldg.
BINKLEY, J. SAM ..... Medical Arts Bldg.
BLACHLEY, C. D. ..... 126 North East 8th St.
BLACHLEY, LUCILLE S., 126 North East 8th St.
BOATRIGHT, LLOYD C. ..... Perrine Bldg.
BONDURANT, C. P. ..... Medical Arts Bldg.
BOLEND, REX ..... Medical Arts Bldg.
BOGGS, NATHAN ..... Perrine Bldg.
BONHAM, WM. L. ..... Medical Arts Bldg.
BORECKY, GEORGE L. ..... Ramsey Tower
BOWEN, RALPH ..... 1200 North Walker
BRADLEY, H. C. ..... Perrine Bldg.
BRANHAM, D. W. ..... 301 West Twelfth St.
BREWER, A. M. ..... Perrine Bldg.
BROWN, G. W. ..... Medical Arts Bldg.
BRUNDAGE, C. L. ..... 1200 North Walker
BUCHANAN, THOS. A., American Natl. Bank Bldg.
BURTON, JOHN F. ..... Osler Bldg.
BUTLER, H. W. ..... 1200 North Walker
CAILEY, LEO F. ..... Medical Arts Bldg.
CAMPBELL, COYNE H. ..... Medical Arts Bldg.
CANADA, J. C. ..... 216½ West Commerce
CATES, ALBERT ..... Medical Arts Bldg.
CAVINESS, J J. ..... Medical Arts Bldg.
CHARNEY, L. H. ..... Medical Arts Bldg.
CHASE, A. B. ..... Colcord Bldg.
CLARK, ANSON L. ..... Medical Arts Bldg.
CLOUDMAN, H. H. ..... Medical Arts Bldg.
CLYMER, CYRIL E. ..... Medical Arts Bldg.
COLEY, A. J. ..... Hightower Bldg.
COLLINS, H. D. ..... Medical Arts Bldg.
COOPER, F. M. ..... Medical Arts Bldg.
CUNNINGHAM, S. R. ..... Medical Arts Bldg.
DAILY, H. J. ..... Medical Arts Bldg.
DICKSON, GREEN K. ..... 1200 North Walker
DAVIS, C. E. ..... Medical Arts Bldg.
DAVIS, E. P. ..... 918 North West 23rd St.
DEMAND, F. A. ..... 1200 North Walker
DERSCH, WALTER H. ..... Medical Arts Bldg.
DILL, FRANCIS E. ..... Medical Arts Bldg.
DOWDY, THOS. W. ..... Medical Arts Bldg.
DOUGHERTY, VIRGIL T., Gorei, Abyssinia, Africa
DUNCAN, D. G ..... Medical Arts Bldg.
EARLY, R. O. ..... Medical Arts Bldg.
EASTLAND, WM. E. ..... Medical Arts Bldg.
ELEY, N. P. ..... 400 West Tenth St.
EMENHEISER, LEE K. ..... Washington University, St. Louis, Mo.
EPLEY, C. O. ..... 418 Osler Bldg.
ESKRIDGE, J. B. ..... 1200 North Walker
ERWIN, F. B. ..... Medical Arts Bldg.
FAGIN, HERMAN ..... 400 West Tenth St.
FARIS, BRUNEL D. ..... Medical Arts Bldg.
FERGUSON, E. G. ..... Medical Arts Bldg.
FERGUSON, E. S. ..... Medical Arts Bldg.
FISHMAN, C. J. ..... 132 West Fourth St.
FITZ, R. G., Taming Fu Hoppi, Prov. No. China
FLESHER, THOS. H. ..... Edmond
FRIERSON, S. E. ..... Medical Arts Bldg.

| | |
|---|---|
| FRYER, S. R. | 119 West Fifth St. |
| FULTON, CLIFFORD C. | Medical Arts Bldg. |
| FULTON, GEO. S. | American Natl. Bank Bldg. |
| GARRISON, GEO. H. | 1200 North Walker |
| GEE, O. J. | Medical Arts Bldg. |
| GIBBS, ALLEN G. | Ramsey Tower |
| GLOMSET, JOHN L. | 621 Osler Bldg. |
| GOLDFAIN, E. | 717 North Robinson |
| GOODWIN, R. Q. | Medical Arts Bldg. |
| GRAHAM, ALLISON T. | 104 West Commerce |
| GRAY, J. WORTH | 1315 South Agnew |
| GUTHRIE, A. L. | 1200 North Walker |
| GREGORY, M. S. | Medical Arts Bldg. |
| HALL, CLARK H. | Medical Arts Bldg. |
| HANEY, A. H. | Perrine Bldg. |
| HAMMONDS, O. O. | 623 North East 18th St. |
| HARBISON, FRANK | Terminal Bldg. |
| HARBISON, J. E. | Terminal Bldg. |
| HARRIS, H. W. | 1200 North Walker |
| HASKETT, PAUL E. | Hales Bldg. |
| HAYES, BASIL A. | Osler Bldg. |
| HATCHETT, J. A. | Medical Arts Bldg. |
| HAZEL, O. G. | Medical Arts Bldg. |
| HEATLEY, JOHN E. | Medical Arts Bldg. |
| HERMANN, JESS | Medical Arts Bldg. |
| HETHERINGTON, A. J. | 2014 Gatewood Ave. |
| HICKS, F. B. | Medical Arts Bldg. |
| HIRSCHFIELD, A. C. | Medical Arts Bldg. |
| HOLLIDAY, J. R. | 1200 North Walker |
| HOOD, F. REDDING | 1200 North Walker |
| HOWARD, ROBT. L. | 1200 North Walker |
| HOWARD, R M. | Osler Bldg. |
| HUNTER, GEO. W. | Wewoka |
| HUGGINS, J. R. | Box 127, Clinton |
| HYROOP, GILBERT L. | Medical Arts Bldg. |
| JACKSON, A. R. | 2528½ South Robinson |
| JACOBS, MINARD F. | Medical Arts Bldg. |
| JANCO, LEON | 10 West Park Place |
| JETER, HUGH | 1200 North Walker |
| JOBE, VIRGIL R. | Capitol Bldg. |
| JOLLY, W. J. | 614 West 14th St. |
| JONES, HUGH C. | Medical Arts Bldg. |
| KAHLE, C. E. | 1738 North West 16th St. |
| KELLER, W. F. | Medical Arts Bldg. |
| KELSO, J. | Medical Arts Bldg. |
| KELTZ, BERT F. | Medical Arts Bldg. |
| KERNODLE, S. E. | First Natl. Bldg. |
| KIMBALL, G. H. | Ramsey Tower |
| KIMBALL, MELVIN | Medical Arts Bldg. |
| KUHN, JOHN F. | Medical Arts Bldg. |
| KUHN, Jr., JOHN F. | Medical Arts Bldg. |
| LAIN, E. S. | Medical Arts Bldg. |
| LAMB, JOHN H. | Medical Arts Bldg. |
| LAMBKE, PHIL M. | 1220 South East 29th St. |
| LA MOTTE, GEORGE A. | Colcord Bldg. |
| LANE, L. C. | 311½ North West Ninth St. |
| LANGSFORD, WM. | 328 East Eleventh St. |
| LANGSTON, WANN | Medical Arts Bldg. |
| LAWSON, N. E. | 1616 West 26th St. |
| LAWSON, PAT | Medical Arts Bldg. |
| LEE, CLARENCE | Hightower Bldg. |
| LENEY, FANNIE LOU | 400 West Tenth St. |
| LEMON, CECIL W. | Medical Arts Bldg. |
| LEONARD, C. E. | Medical Arts Bldg. |
| LEWIS, A. R. | Hightower Bldg. |
| LINGENFELTER, F. M. | 1200 North Walker |
| LITTLE, JOHN R. | Ramsey Tower |
| LONG, LEROY | Medical Arts Bldg. |
| LONG, LEROY D. | Medical Arts Bldg. |
| LONG, ROSS D. | 617 West 14th St. |
| LONG, WENDELL | Medical Arts Bldg. |
| LOVE, R. S. | Perrine Bldg. |
| LOWRY, DICK | 1200 North Walker |
| LOWRY, TOM | 1200 North Walker |
| LOY, C. F. | Perrine Bldg. |
| LYON, J. I. | Edmond |
| MacCABE, R. S. | Medical Arts Bldg. |
| MacDONALD, J. C. | 300 West Twelfth St. |
| MARGO, E. | 717 North Robinson St. |
| MARTIN, J. T. | 1200 North Walker |
| MARTIN, HOWARD | Ramsey Tower |
| McBRIDE, E. D. | 717 North Robinson |
| McGEE, J. P. | 404 Osler Bldg. |
| McHENRY, D. D. | 508 North West 15th St. |
| McHENRY, L. C. | Medical Arts Bldg. |
| McKINNEY, MILAN | Medical Arts Bldg. |
| McLAUGHLIN, J. R. | Medical Arts Bldg. |
| McNEILL, PHIL M. | Medical Arts Bldg. |
| MECHLING, GEO. S. | Osler Bldg. |
| MESSENBAUGH, J. F. | Colcord Bldg. |
| MILES, W. H. | City Hall Bldg. |
| MILLER, N. L. | Medical Arts Bldg. |
| MILLS, R. C. | City Hall Bldg. |
| MOFFITT, JOHN A. | University Hospital |
| MOORE, ELLIS | Medical Arts Bldg. |
| MOORE, C. D. | Perrine Bldg. |
| MOOR, H. D. | 800 East Thirteenth St. |
| MOORMAN, FLOYD | 1200 North Walker |
| MOORMAN, L. J. | 1200 North Walker |
| MORGAN, C. A. | First Natl. Bldg. |
| MORLEDGE, WALKER | Osler Bldg. |
| MORRISON, H. C. | 807 North West 23rd St. |
| MOTH, M. V. | American Bldg. |
| MULVEY, BERT E. | Medical Arts Bldg. |
| MURDOCH, R. L. | Medical Arts Bldg. |
| MUSICK, ELMER R. | Medical Arts Bldg. |
| MUSICK, V. H. | 217½ West Commerce |
| MUSSIL, W. M. | Medical Arts Bldg. |
| MYERS, RALPH | 1200 North Walker |
| NAGLE, PATRICK | Medical Arts Bldg. |
| NICHOLSON, B. H. | 300 West Twelfth St. |
| NIELSEN, GERTRUDE | 1200 North Walker |
| O'DONOGHUE, D. H. | Medical Arts Bldg. |
| PADBERG, J. W. | 1800 West 16th St. |
| PATTERSON, ROBT. U. | 520 North West 34th St. |
| PAULUS, D. D. | 301 West Twelfth St. |
| PENICK, GRIDER | Colcord Bldg. |
| PHELPS, A. S. | Medical Arts Bldg. |
| PINE, JOHN S. | Medical Arts Bldg. |
| POINTS, BLAIR | Luther |
| POSTELLE, J. M. | Medical Arts Bldg. |
| POUNDERS, CARROLL M. | 1200 North Walker |
| PRICE, J. S. | 1200 North Walker |
| RECK, J. A. | Colcord Bldg. |
| REED, EMIL P. | 1200 North Walker |
| REED, HORACE | Osler Bldg. |
| REED, JAMES ROBERT | Medical Arts Bldg. |
| REICHMAN, RUTH S. | 124 North West 15th St. |
| RIELY, LEA A. | Medical Arts Bldg. |
| RILEY, J. W. | 119 West Fifth St. |
| ROBINSON, J. H. | 301 West Twelfth St. |
| RODDY, JOHN A. | 1117 Ramsey Tower |
| RODDY, JOHN A. | Ramsey Tower |
| ROSENBERGER, F. E. | Perrine Bldg. |
| ROUNTREE, C. R. | 1200 North Walker |
| RUCKS, Jr., W. W. | 300 West Twelfth St. |
| RUCKS, W. W. | 300 West Twelfth St. |
| RUHL, A. M. | Edmond |
| SALOMON, A. L. | 1200 North Walker |
| SADLER, LEROY H. | Osler Bldg. |
| SANDS, A. J. | Choctaw |
| SANGER, WINNIE M. | Key Bldg. |
| SANGER, F. M. | Key Bldg. |
| SANGER, F. A. | Key Bldg. |
| SERVER, MILTON | 1200 North Walker |
| SEWELL, DAN R. | 400 North West Tenth St. |
| SEYLER, L. EVERETT | 1200 North Walker |
| SHELTON, J. W. | Medical Arts Bldg. |
| SHEPPARD, MARY V. S. | 1200 North Walker |
| SMITH, CHAS. A. | Medical Arts Bldg. |
| SMITH, D. G. | Medical Arts Bldg. |
| SMITH, WENDELL L. | Medical Arts Bldg. |
| SNOW, J. B. | 1200 North Walker |
| STARRY, L. J. | 1200 North Walker |
| STILWELL, ROBT. J. | American Natl. Bldg. |
| STONE, S. N. | Edmond |

STOUT, M. E. ..... 209 West Thirteenth St.
STRADER, S. E. ..... Hightower Bldg.
STANBRO, G. E. ..... Medical Arts Bldg.
STROTHER, S. P. ..... 120 North West 23rd St.
SULLIVAN, ELIJAH S. ..... Medical Arts Bldg.
SULLIVAVN, ERNEST ..... Hightower Bldg.
TABOR, GEO. R. ..... First National Bank
TAYLOR, C. B. ..... Medical Arts Bldg.
TAYLOR, W. M. ..... 1200 North Walker
THOMPSON, W. J. ..... Osler Bldg.
TODD, H. C. ..... Colcord Bldg.
TOOL, DONOVAN ..... Edmond
TOWNSEND, CARY ..... Medical Arts Bldg.
TRICE, S. T. ..... 943 North West Thirteenth St.
TURNER, HENRY H. ..... 1200 North Walker
UNDERWOOD, E. L. ..... Hales Bldg.
VALBERG, ERNEST ..... Perrine Bldg.
WAILS, T. G. ..... Medical Arts Bldg.
WARMACK, J. C. ..... 200 North West 16th St.
WATSON, O. ALTON ..... 400 West Tenth St.
WATSON, R. D. ..... Britton
WEIR, MARSHALL W. ..... Ramsey Tower
WEDEL, CURT VON ..... 610 North West Ninth St.
WELLS, EVA ..... Medical Arts Bldg.
WELLS, WALTER A. ..... Medical Arts Bldg.
WEST, W. K. ..... 1200 North Walker
WESTFALL, L. M. ..... Medical Arts Bldg.
WHITE, ARTHUR W. ..... Medical Arts Bldg.
WHITE, OSCAR P. ..... 1200 North Walkker
WHITE, PHIL ..... Perrine Bldg.
WILDMAN, S. F. ..... Medical Arts Bldg.
WILKINS, HARRY ..... Medical Arts Bidg.
WILLIAMS, L. C. ..... Osler Bldg.
WILLIAMSON, W. H. ..... First Natl. Bldg.
WILSON, E. C. ..... 1001 NorthEast 20th St.
WILSON, KKENNETH J. ..... Medical Arts Bldg.
WOLFF, J. P. ..... Osler Bldg.
WOODWARD, NEIL W. ..... 1200 North Walker
WRIGHT, HARPER ..... 240 West Commerce
YEAKEL, E. L. ..... 406 Savings Bldg.

## OKMULGEE

ALEXANDER, LIN ..... Okmulgee
ALEXANDER, ROBT. L. ..... Okmulgee
ALEXANDER, T. C., C.C.C. Hdq., Phoenix, Ariz.
BOLLINGER, I. W. ..... Henryetta
BOSWELL, H. D. ..... Henryetta
CARLOSS, T. C. ..... Morris
CARNELL, M. D. ..... Okmulgee
CONN, L. D. ..... Box 186, Del Rio, Texas
COTTERAL, J. R. ..... Okmulgee
*COTT, W. M. ..... Okmulgee
EDWARDS, J. G. ..... Okmulgee
GLISMANN, M. B. ..... Okmulgee
HOLMES, A. R. ..... Henryetta
KILPATRICK, G. A. ..... Henryetta
LESLIE, S. B. ..... Okmulgee
MABEN, CHAS. S. ..... Okmulgee
MATHENEY, J. C. ..... Okmulgee
McKINNEY, G. Y. ..... Henryetta
MING, C. M. ..... Okmulgee
MITCHENER, W. C. ..... Okmulgee
NELSON, J. P. ..... Beggs
RAINS, H. L. ..... Okmulgee
RANDEL, D. M. ..... Okmulgee
RANDELL, H. O. ..... Okmulgee
*REMBERT, J. C. ..... Okmulgee
ROBINSON, J. C. ..... Henryetta
RODDA, E. D. ..... Okmulgee
SIMPSON, N. N. ..... Henryetta
STARK, W. W. ..... 717 N. Robinson, Oklahoma City
TORRANCE, L. B. ..... Okmulgee
VERNON, W. C. ..... Okmulgee
WALLACE, V M. ..... Morris
WATSON, F. S. ..... Okmulgee
WATSON, W. S. ..... Okmulgee
*WIDENER, DEAN C. ..... Okmulgee

## OSAGE

AARON, W. H. ..... Pawhuska
BAYLOR, R. A. ..... Fairfax
CATON, C. N. ..... Wynona
DALY, J. F. ..... Pawhuska
DOZIER, B. E. ..... Snidler
GOVAN, T. P. ..... Pawhuska
GUILD, C. H. ..... Shidler
HEMPHILL, G. K. ..... Pawhuska
HEMPHILL, P. H. ..... Pawhuska
KARASEK, M. ..... Shidler
KEYES, E. C. ..... Shidler
LIPE, E. N. ..... Fairfax
LOGAN, C. K. ..... Hominy
RAGAN, T. A. ..... Fairfax
RUST, M. E. ..... Pawhuska
SMITH, R. O. ..... Hominy
SULLIVAN, B. F. ..... Barnsdall
SUMMERS, H. L. ..... Marion, Ill.
WALKER, G. I. ..... Hominy
WALKER, ROSCOE ..... Pawnuska
WEIRICH, C. R. ..... Pawhuska
WORTEN, D. ..... Pawhuska

## OTTAWA

AISENSTADT, E. ALBERT ..... Picher
BARRY, J. R. ..... Picher
BUTLER, V. V. ..... Picher
CANNON, R. F. ..... Miami
CHESNUT, W. G. ..... Miami
COLVERT, G. W. ..... Miami
CONNELL, MATT ..... Picher
COOTER, A. M. ..... Miami
CRAIG, J. W. ..... Miami
CUNNINGHAM, P. J. ..... Miami
DeARMAN, M. M. ..... Miami
DeTAR, G. A. ..... Miami
DOLAN, W. M. ..... Picher
GRAY, C. M. ..... Miami
HAMPTON, J. B. ..... Commerce
HUGHES, A. R. ..... Wyandotte
JACOBS, J. C. ..... Miami
JACOBY, J. S. ..... Commerce
KERR, W. C. ..... Picher
KITCHEN, J. C. ..... Afton
LIGHTFOOT, J. B. ..... Miami
McNAUGHTON, G. P. ..... Miami
MILLER, H. K. ..... Fairland
PHILLIPS, ISAAC ..... Picher
PINNELL, GENERAL ..... Miami
RALSTON, B. W. ..... Commerce
RITCHEY, H. C. ..... Picher
RUSSELL, RICHARD ..... Picher
SHELTON, B. W. ..... Miami
SMITH, W. B. ..... Fairland
WORMINGTON, F. L. ..... Miami

## PAWNEE

BEITMEN, C. E. ..... Kendrick
BROWNING, R. L. ..... Pawnee
CARMICHAEL, M. M. ..... Jennings
HETHERINGTON, L. P. ..... Pawnee
JONES, R. E. ..... Pawnee
LeHEW, ELTON W. ..... Pawnee
LeHEW, J. L. ..... Pawnee
ROBINSON, E. T. ..... Cleveland
SADDORIS, M. L. ..... Cleveland
SPALDING, H. B. ..... Ralston

*Deceased

## PAYNE

ADAMS, JAMES E. ..... Cushing
BASSETT, C. M. ..... Cushing
BEACH, C. H. ..... Glencoe
CLEVERDON, L. A. ..... Stillwater
DAVIS, BENJ. ..... Cushing
FRIEDMANN, P. W. ..... 5842 Harper Ave., Chicago
FRY, POWELL E. ..... Stillwater
HARRIS, E. M. ..... Cushing
HERRINGTON, D. J ..... Cushing
HOLBROOK, R. W. ..... Perkins
LEATHEROCK, R. E. ..... Cushing
LOVE, T. A. ..... Ripley
MANNING, H. C. ..... Cushing
MARTIN, EMMETT O. ..... Cushing
MARTIN, JOHN F. ..... Stillwater
MARTIN, J. W. ..... Cushing
MITCHELL, L. A. ..... Stillwater
PHILLIPS, J. W. ..... Oilton
RICHARDSON, P. M. ..... Cushing
ROBERTS, R. E. ..... Stillwater
SMITH, A. B. ..... Stillwater
STRAHN, EVA ..... Stillwater
WAGGONER, ROY E. ..... Stillwater
WALTRIP, J. R. ..... Yale
WILHITE, L. R. ..... Perkins

## PITTSBURG

BARTHELD, F. T. ..... McAlester
BAUM, F. J. ..... McAlester
BRIGHT, J. B. ..... Kiowa
BRONSON, C. J. ..... McAlester
BUNN, A. D. ..... Savanna
BUSSEY, H. N., 2043 West 23rd St., Oklahoma City
DORROUGH, JOE ..... McAlester
GEORGE, L. J. ..... Stuart
GREENBERGER, E. D. ..... McAlester
HAILEY, W. P. ..... Haileyville
HARRIS, C. T. ..... Kiowa
KIES, B. B. ..... McAlester
KILPATRICK, G. A. ..... McAlester
KLOTZ, WM. F. ..... McAlester
KUYRKENDALL, L. C. ..... McAlester
LIVELY, CLAUDE E. ..... McAlester
McCARLEY, T. H. ..... McAlester
MILLER, F. A. ..... Hartshorne
MILLS, CHAS. K. ..... McAlester
MUNN, J. A. ..... McAlester
NORRIS, T. T. ..... Krebs
PALMER, CLARA ..... McAlester
PARK, J. F. ..... McAlester
PEARCE, C. M., State Capitol Bldg., Oklahoma City
PEMBERTON, R. K. ..... McAlester
RAMSEY, W. G. ..... Quinton
RICE, O. W. ..... McAlester
RUSSELL, ALLEN R. ..... McAlester
SAMES, W. W. ..... Hartshorne
SHULLER, E. H. ..... McAlester
THOMAS, ERNEST ..... Quinton
WELCH, A. J. ..... McAlester
WILLIAMS, C. O. ..... McAlester
WILLOUR, L. S. ..... McAlester
WILSON, HERBERT A. ..... McAlester
WILSON, McCLELLAND ..... McAlester

## PONTOTOC

BRECO, DAVIS ..... Ada
BRECO, J. G. ..... Ada
BRYDIA, CATHERINE ..... Ada
BURROWS, L. I. ..... Ada
CANADA, E. A. ..... Ada
CRAIG, JOHN R. ..... Ada
CUMMINGS, ISHAM L. ..... Ada
DAWSON, B. B. ..... Ada
DEAN, W. F. ..... Ada
GULLATT, E. M. ..... Ada
HINES, SIDNEY, J. T. ..... Fittstown
HOLLOWAY, THOS. R. ..... Stonewall
KING, RICHARD F. ..... Ada
LANE, WILSON H. ..... Ada
LEWIS, E. F. ..... Ada
LEWIS, MILES L. ..... Ada
McKEEL, SAM A. ..... Ada
McNEW, M. C. ..... Ada
MILLER, OSCAR H. ..... Ada
MOREY, JOHN B. ..... Ada
NEEDHAM, C. F. ..... Ada
PAULSON, GEORGE A. ..... Ada
PETERSON, WM. G. ..... Ada
ROSS, SAMUEL P. ..... Ada
RUTLEDGE, JAS. A. ..... Ada
SEABORN, T. L. ..... Roff
SUGG, ALFRED R. ..... Ada
THRELKELD, WM. R. ..... Ada
WEBSTER, M. M. ..... Ada
WELBORN, ORANGE E. ..... Ada

## POTTAWATOMIE

ANDERSON, R. M. ..... Shawnee
APPLEWHITE, G. H. ..... Shawnee
BAKER, M. A. ..... Shawnee
BALL, W. A. ..... Wanette
BAXTER, G. S. ..... Shawnee
BLOUNT, W. T. ..... Maud
BROWN, R. A. ..... Prague
BYRUM, J. M. ..... Shawnee
CAMPBELL, H. G. ..... St. Louis, Okla.
CARSON, F. L. ..... Shawnee
CORDELL, U. S. ..... McComb
CULBERTSON, R. R. ..... Maud
CULLUM, J. E. ..... Earlsboro
FORTSON, J. L. ..... Tecumseh
GALLAHER, F. C. ..... Shawnee
GALLAHER, W. M. ..... Shawnee
GASTON, JOHN I. ..... Shawnee
GILLICK, D. W. ..... Shawnee
HUGHES, H. E. ..... Shawnee
HUGHES, J. E. ..... Shawnee
KAYLOR, R. C. ..... McCloud
KEEN, FRANK M. ..... Shawnee
MATTHEWS, W. F. ..... Tecumseh
McFARLING, A. C. ..... Shawnee
McADAMS, WM. ALPHA ..... Shawnee
NEWLIN, FRANCES P. ..... Shawnee
PARAMORE, C. F. ..... Shawnee
RICE, E. E. ..... Shawnee
ROWLAND, T. D. ..... Shawnee
ROYSTER, J. H. ..... Wanette
STEVENS, WALTER S., 315 P. O. Bldg., Okla. City
STOOKSBURY, J. M. ..... Shawnee
*WAGNER, H. A. ..... Shawnee
WALKER, J. A. ..... Shawnee
*WALKER, J. E. ..... Shawnee

## PUSHMATAHA

BURNETT, J. A. ..... Waldron, Ark.
CONNALLY, D. W. ..... Antlers
GUINN, E. ..... Antlers
HUCKABAY, B. M. ..... Greenville, Tex.
JOHNSON, H. C. ..... Antlers
KIRKPATRICK, J. ..... Tuskahoma
LAWSON, JOHN S. ..... Antlers
PATTERSON, E. S. ..... Antlers

## ROGERS

ANDERSON, F. A. ..... Claremore
ARNOLD, A. M. ..... Claremore
BASSMAN, CAROLINE ..... Claremore
BESON, C. W. ..... Claremore

*Deceased

| | |
|---|---|
| BUSHYHEAD, J. C. | Claremore |
| COLLINS, B. F. | Claremore |
| HAYS, W. F. | Claremore |
| HOWARD, W. A. | Chelsea |
| JENNINGS, K. D. | Chelsea |
| MASON, W. S. | Claremore |
| MELOY, R. C. | Claremore |
| NELSON, D. C. | Claremore |

SEMINOLE

| | |
|---|---|
| BRIGGS, T. H. | Wewoka |
| CHAMBERS, C. S. | Seminole |
| DEATON, A. N. | Wewoka |
| GIESEN, A. F. | Konawa |
| GRIMES, J. P. | Wewoka |
| HACKLER, J. F. | Seminole |
| HARBOR, J. N. | Seminole |
| HARRISON, T. F. | Wewoka |
| HARTSHORNE, W. O. | Cromwell |
| KNIGHT, W. L. | Wewoka |
| MARTIN, W. S. | Wewoka |
| McGOVERN, J. D. | Wewoka |
| MILLS, N. W. | Snomac |
| MOSHER, D. D. | Seminole |
| PACE, L. R. | Seminole |
| PIERSON, D. D. | Konowa |
| SCOTT, T. A. | Bowlegs |
| STEPHENS, A. B. | Seminole |
| THROGMORTON, H. B. | Seminole |
| TURLINGTON, M. M. | Seminole |
| VAN SANDT, G. B. | Wewoka |
| VAN SANDT, M. M. | Wewoka |
| WALKER, A. A. | Wewoka |
| WARE, T. H. | Seminole |

SEQUOYAH

| | |
|---|---|
| MORROW, J. A. | Sallisaw |

STEPHENS

| | |
|---|---|
| BREWER, J. R. | Marlow |
| BURNETT, B. H. | Duncan |
| CARMICHAEL, J. B. | Duncan |
| DOWNING, G. G. | Marlow |
| HARRISON, C. M. | Comanche |
| IVY, W. S. | Duncan |
| KING, E. G. | Duncan |
| LINDLEY, E. C. | Duncan |
| LINZY, J. H. | Comanche |
| *LONG, D. | Duncan |
| McCLAIN, W. Z. | Marlow |
| McMAHAN, A. M. | Duncan |
| NIEWEG, J. W. | Route 4, Shawnee |
| OVERTON, L. M. | Shawnee |
| PATTERSON, J. L. | Duncan |
| PRUITT, C. C. | Comanche |
| RICHARDSON, R. W. | Duncan |
| SALMON, W. T. | Duncan |
| SMITH, L. P. | Marlow |
| TALLEY, C. N. | Marlow |
| THOMASON, E. B. | Marlow |
| WEEDN, A. J. | Duncan |
| WILLIAMSON, S. H. | Duncan |

TEXAS

| | |
|---|---|
| HAYES, R. B. | Guymon |
| LEE, DANIEL S. | Guymon |
| REED, PAUL | Texhoma |
| SMITH, MORRIS | Guymon |
| THURSTON, HARRY E. | Texhoma |

TILLMAN

| | |
|---|---|
| ALLEN, C. C. | Frederick |
| ARRINGTON, J. E. | Frederick |
| BACON, O. G. | Frederick |
| CHILDERS, J. E. | Tipton |
| DAVIS, W. | Davidson |
| FISHER, R. L. | Frederick |
| FUQUE, W. A. | Grandfield |
| OSBORN, JR., JAMES D. | Frederick |
| REYNOLDS, J. C. | Frederick |
| SPURGEON, T. F. | Frederick |

TULSA

| | |
|---|---|
| ALLEN, V. K. | 1001 Medical Arts Bldg. |
| ALLISON, T. P. | Sand Springs |
| ARMSTRONG, O. C. | 912 Medical Arts Bldg. |
| AMENT, C. M. | 305 Ritz Bldg. |
| ATCHLEY, R. Q. | 507 Medical Arts Bldg. |
| ATKINS, PAUL N. | 1011 Medical Arts Bldg. |
| BARHAM, J. H. | 314 New Daniel Bldg. |
| BEESLEY, W. W. | 501 Medical Arts Bldg. |
| BEYER, J. W. | 501 Palace Bldg. |
| BILLINGTON, J. J. | 404 Medical Arts Bldg. |
| BLACK, HAROLD J. | 209 Medical Arts Bldg. |
| BOLTON, J. F. | 211 Medical Arts Bldg. |
| BRADFIELD, S. J. | 607 Medical Arts Bldg. |
| BRADLEY, C. E. | 202 Medical Arts Bldg. |
| BRANLEY, BERNARD L. | 315 Medical Arts Bldg. |
| BRASWELL, JAS. C. | 1109 Medical Arts Bldg. |
| BROGDEN, J. C. | 414-15 Medical Arts Bldg. |
| BROOKSHIRE, J. E. | 313 Ritz Bldg. |
| BROWN, H. S. | 615 Medical Arts Bldg. |
| BRYAN, Jr., W. J. | 8 Medical Arts Bldg. |
| CALHOUN, C. E. | Sand Springs |
| CALHOUN, WALTER H. | 1010 Daniels Bldg. |
| CALLAHAN, H. W. | 902 Medical Arts Bldg. |
| CAMPBELL, W. M. | 1301½ East 15th St. |
| CARNEY, A. B. | Atlas Life Bldg |
| CHALMERS, J. S. | Sand Springs |
| CHARBONNET, P. N. | Medical Arts Bldg. |
| CHILDS, D. B. | 1226 South Boston |
| CHILDS, HENRY C. | 1226 South Boston |
| CHILDS, J. W. | 1226 South Boston |
| CLINTON, FRED S. | 823 Wright Bldg. |
| COHENOUR, E. L. | 1102 Medical Arts Bldg. |
| CLULOW, GEO. H. | 410 McBirney Bldg. |
| COOK, ALBERT W. | 1006 Medical Arts Bldg. |
| COULTER, T. B. | 1011 Medical Arts Bldg. |
| CRAWFORD, WM. S. | 1732 South Yorktown |
| CRONK, FRED Y. | 801-05 Medical Arts Bldg. |
| DAILY, R. E. | Bixby |
| DAVIS, A. H. | 710 Medical Arts Bldg. |
| DAVIS, T. H. | 404 Medical Arts Bldg. |
| DEAN, W. A. | 610 Medical Arts Bldg. |
| DENNY, E. R. | 1105 Medical Arts Bldg. |
| DIEFFENBACH, N. J. | 704 Medical Arts Bldg. |
| DILLON, C. A. | 212 Daniel Bldg. |
| DUNLAP, ROY W. | 808 Medical Arts Bldg. |
| EDWARDS, D. L. | 1109 Medical Arts Bldg. |
| EMERSON, A. V. | 212 Medical Arts Bldg. |
| EADS, CHAS H. | 607 Medical Arts Bldg. |
| FARRIS, H. LEE | 303 Medical Arts Bldg. |
| FLACK, F. L. | Natl. Bank of Tulsa Bldg. |
| FLANAGAN, O. A. | 213 Medical Arts Bldg. |
| FORD, H. W. | 417 Oklahoma Natl. Bank Bldg. |
| FULCHER, JOSEPH | 417 Medical Arts Bldg. |
| GARABEDIAN, G. | 1235 South Boulder Ave. |
| GARRETT, D. L. | 701 Medical Arts Bldg. |
| *GEISSLER, P. C. | 1643 Evanston St. |
| GILBERT, J. B. | Roberts Bldg. |
| GLASS, FRED A. | 404 Medical Arts Bldg. |
| GODDARD, R. K. | Skiatook |
| GOODMAN, SAMUEL | 603 Medical Arts Bldg. |
| GORRELL, J. FRANKLIN, | 610 Medical Arts Bldg. |
| GRAHAM, H. C. | 1501 South Baltimore |
| GREEN, HARRY | 1116 Medical Arts Bldg. |
| GROSSHART, PAUL | 302 Medical Arts Bldg. |
| HALL, G. H. | 427 McBirney Bldg. |
| HARALSON, CHAS. H. | 816 Medical Arts Bldg. |

*Deceased

HARRIS, BUNN, Box 356, Jenks
HART, M. M. 1232 South Boulder
HART, M. O. 1232 South Boulder
HASKINS, THOS. M. 814 Daniels Bldg.
HAYS, LAVERNE 1001 Medical Arts Bldg.
HENDERSON, F. W. 304 Medical Arts Bldg.
HENLEY, M. D. 911 Medical Arts Bldg.
HENRY, D. H. 801 Medical Arts Bldg.
HENRY, G. H. 801 Medical Arts Bldg.
HOKE, C. C. 207 Philtower Bldg.
HOOPER, J. S. 518 West Fourth St.
HOOVER, WILKIE D. 320 Philcade Bldg.
HOTZ, CARL J. 604 South Cincinnati
HOUSER, M. A. 606 Beacon Life Bldg.
HUBER, WALTER A. 1113-14 Medical Arts Bldg.
HUDSON, MARGARET G., 411 Medical Arts Bldg.
HUDSON, DAVID V. 214 Medical Arts Bldg.
HUMPHREY, B. H. Sperry
HUTCHISON, A. Bixby
HYATT, EMRY G. 604 South Cincinnati
JACKSON, L. T. 206½ South Main
JOHNSON, CHAS. D. 1117 Medical Arts Bldg.
JOHNSON, R. R. Sand Springs
JONES, W. M. 204 Medical Arts Bldg.
KEMMERLY, H. P. 902 Medical Arts Bldg.
KRAMER, A. C. 415 Medical Arts Bldg.
LARRABEE, W. S. 411 Medical Arts Bldg.
LAWS, J. H. Broken Arrow
LEE, J. K. 210 Medical Arts Bldg.
LeMASTER, D. W. 902 Medical Arts Bldg.
LHEVINE, MORRIS B. 1007 Medical Arts Bldg.
LONEY, W. R. R. 301 Medical Arts Bldg.
LYNCH, T. J. Philcade Bldg
LOWE, J. O. Atlas Bldg.
MacKENZIE, IAN 510 Medical Arts Bldg.
MARGOLIN, B. 210 Medical Arts Bldg.
MAYGINNIS, P. H. 505 Palace Bldg.
McCOMB, L. A. 801 Medical Arts Bldg.
McDONALD, D. M. 24 East 17th St.
McDONALD, J. E. Medical Arts Bldg.
McGILL, RALPH A. 1010 Medical Arts Bldg.
McGUIRE, H. J. 910 Medical Arts Bldg.
McKELLAR, MALCOLM 604 South Cincinnati
McLEAN, B. W. Jenks
McQUAKER, MOLLY 1648 East 13th St.
MILLER, GEORGE H. 206 Atlas Bldg.
MINER, J. L. 810 Medical Arts Bldg.
MISHLER, DONALD L. 604 South Cincinnati
MOHRMAN, SYLVESTER, 611 New Daniels Bldg.
MUNDING, L. A. Medical Arts Bldg.
MURDOCK, H. D. 1011 Medical Arts Bldg.
MURRAY, P. G. 506 Medical Arts Bldg.
MURRAY, SILAS 501 Medical Arts Bldg.
MYERS, F. C. 502 Daniels Bldg.
NAPPER, MARVIN L. Medical Arts Bldg.
NAUHEIM, H. S. 511 Medical Arts Bldg.
NEAL, JAMES H. 1944 North Denver Place
NELSON, F. J. 603 Medical Arts Bldg.
NELSON, F. L. 614 Daniels Bldg.
NELSON, I. A. 1107 Medical Arts Bldg.
NELSON, M. O. 307 Medical Arts Bldg.
NESBITT, E. P. 917 Medical Arts Bldg.
NESBITT, P. P. 917 Medical Arts Bldg.
NORMAN, G. R. 17½ North Lewis
NORTHRUP, L. C. 410 McBirney Bldg.
OSBORN, GEO. R. 1105 Medical Arts Bldg.
PAYY, C. A. 801 Medical Arts Bldg.
PEDEN, J. C. 612 Medical Arts Bldg.
PERRY, HUGH, 416 McBirney Bldg.
PIGFORD, A. W. 1001 Medical Arts Bldg.
PIGFORD, R. C. 1001 Medical Arts Bldg.
PORTER, H. H. 510 Medical Arts Bldg.
PRESSON, L. C. 902 Medical Arts Bldg.
PRICE, HARRY 407 Medical Arts Bldg.
RAY, R. G. 402 Atlas Bldg.
REESE, K. C. 1101 Medical Arts Bldg.
REYNOLDS, J. L. 305 Palace Bldg.
RHODES, R. E. LEE 509 Medical Arts Bldg.
RICHEY, S. M. 1304½ West 17th St.
ROBERTS, T. R. 425 Wright Bldg.
ROGERS, J. W. 407 Medical Arts Bldg.
ROTH, A. W. 607 Medical Arts Bldg.
ROY, EMILE 317 Wright Bldg.
RUPRECHT, H. A. 604 South Cincinnati
RUPRECHT, MARCELIA 604 South Cincinnati
RUSHING, F. E. 505 Medical Arts Bldg.
RUSSELL, G. R. 604 South Cincinnati
SEARLE, M. J. 501 Medical Arts Bldg.
SHEPARD, R. M. 306 Medical Arts Bldg.
SHEPARD, S. C. 706 Medical Arts Bldg.
SHERWOOD, R. G. 412 Wright Bldg.
SHOWMAN, W. A. 409 Medical Arts Bldg.
SHIPP, J. D. Sisler Clinic
SIMPSON, CARL F. 502 Medical Arts Bldg.
SINCLAIR, F. D. Springer Clinic
SIPPLE, M. E. 1411½ South Troost
SISLER, WADE 807 South Elgin
SMITH, D. O. 604 South Cincinnati St.
SMITH, JOHN H., 204 Isham Bldg., Longview, Texas
SMITH, N. R. 703 Medical Arts Bldg.
SMITH, ROY L. 711 Medical Arts Bldg.
SMITH, R. N. 1017 Medical Arts Bldg.
SMITH, RONALD R. 403 Daniels Bldg.
SMITH, W. O. 323 Philcade Bldg.
SPANN, L. A. 305 Roberts Bldg.
SPRINGER, M. P. 604 South Cincinnati
STALLINGS, T. W. 114 West Fourth St.
STANLEY, MONT V. 710 Daniels Bldg.
STEVENSON, JAS. 615 Medical Arts Bldg.
STEWART, H. B. 1516 East 21st St.
STUART, FRANK A. 311 Medical Arts Bldg.
STUART, L. H. 1107 Medical Arts Bldg.
SUMMERS, C. S. 611 Daniels Bldg.
TRAINOR, W. J. 1011 Medical Arts Bldg.
TUCKER, I. N. 511 Medical Arts Bldg.
UNDERWOOD, D. J. 414-15 Medical Arts Bldg.
UNDERWOOD, F. L. 1001 Medical Arts Bldg.
VENABLE, S. C. 720 Mayo Bldg.
WAINWRIGHT, A. G. 424 McBirney Bldg.
WALKER, W. A. 332 Kennedy Bldg.
WALLACE, J. E. 914 Medical Arts Bldg.
WALL, G. A. 902 Medical Arts Bldg.
WARD, B. W. 823 Wright Bldg.
WHITE, P. C. 312 Medical Arts Bldg.
WHITE, NELSON S. 416 Medical Arts Bldg.
WILEY, A. RAY 812 Medical Arts Bldg.
WILKS, F. M. Route No. 3, Box 262
WITCHER, R. B. 910 Medical Arts Bldg
WOODS, C. J. 511 Medical Arts Bldg.
WOODSON, FRED E. 908 Medical Arts Bldg.
ZINK, G. W. Red Fork
ZINK, H. F. 807 Daniels Bldg.
ZINK, ROY 807 Daniels Bldg.

## WAGONER

BATES, S. R. Wagoner
LEONARD, JOHN D. Wagoner
PLUNKETT, J. H. Wagoner
RIDDLE, H. K Coweta

## WASHINGTON

ATHEY, J. V. Bartlesville
BEECHWOOD, E. E. Bartlesville
CHAMBERLIN, E. M. Bartlesville
CRAWFORD, G. W. Dewey
CRAWFORD, H. G. Bartlesville
CRAWFORD, J. E. Bartlesville
CRAWFORD, T. O. Bartlesville
DORSHEIMER, G. V. Dewey
ETTER, F. S. Bartlesville
GREEN, O. I. Bartlesville
HUDSON, L. D. Dewey
KINGMAN, W. H. Bartlesville
LeBLANC, WM. Ochelata

PARKS, S. M. ..... Bartlesville
REWERTS, F. C. ..... Bartlesville
SHIPMAN, W. H. ..... Bartlesville
SMITH, J. G. ..... Bartlesville
SOMERVILLE, O. S. ..... Bartlesville
STAVER, B. F. ..... Bartlesville
TORREY, J. P. ..... Bartlesville
VANSANT, J. P. ..... Dewey
WEBER, H C. ..... Bartlesville
WEBER, S. G. ..... Bartlesville
WELLS, C. J. ..... Bartlesville
WILSON, C. C. ..... Care Dr. T. R. Coleman, East Gulf, West Virginia

## WASHITA

BENNETT, D. W. ..... Sentinel
BUNGARDT, A. H. ..... Cordell
HARMES, J. H. ..... Cordell
JONES, J. PAUL ..... Dill
LIVINGSTON, L. G. ..... Cordell
McMURRAY, J. F. ..... Sentinel
NEAL, A. S. ..... Cordell
STOLL, A. A. ..... Foss
SULLIVAN, C. B. ..... Cordell
TRACY, C. M. ..... Sentinel
WEAVER, E. S. ..... Cordell
WEBER, A. ..... Bessie

## WOODS

BENJEGERDES, THEODORE D. ..... Beaver
BILBY, GEO. N. ..... Route 1, Pea Ridge, Ark.
CAMPBELL, JOHN D. ..... Fairview
CLAPPER, E. P. ..... Waynoka
ENSOR, D. B. ..... Hopeton
GRANTHAM, ELIZABETH ..... Alva
HALE, ARTHUR E. ..... Alva
HALL, RAY L. ..... Waynoka
HAMMER, JOHN E. ..... Kiowa, Kans.
HUNT, ISAAC S. ..... Fredom
McGREW, EDWIN A. ..... Beaver
ROGERS, CHAS. L. ..... Carmen
ROYER, CHAS. A. ..... Kiowa, Kans.
SIMON, JOHN F. ..... Alva
SIMON, WM. E. ..... Alva
STEPHENSON, ISHMEL F. ..... Alva
STEPHENSON, WALTER L. ..... Cheyenne
TEMPLIN, OSCAR E. ..... Alva
TRAVERSE, CLIFFORD A. ..... Alva

## WOODWARD

ALFORD, O. T. ..... Seiling
BARBER, J. J. ..... Laverne
BEAM, J. P. ..... Arnett
CAMP, E. F. ..... Buffalo
DARWIN, D. W. ..... Woodward
DAVIS, J. J. ..... Higgins, Texas
DAY, J. L. ..... Supply
DUER, JOE L. ..... Taloga
DUNCAN, J. C. ..... Forgan
FORNEY, C. J. ..... Woodward
HILL, HARRY K. ..... Laverne
JOHNSON, H. L. ..... Supply
LEACHMAN, T. C. ..... Woodward
McMILLAN, J. M. ..... Supply
NEWMAN, FLOYD ..... Shattuck
NEWMAN, MESHECH HASKELL ..... Shattuck
NEWMAN, O. C. ..... Shattuck
NEWMAN, ROY ..... Shattuck
SILVERTHORNE, C. R. ..... Woodward
TEDROWE, C. W. ..... Woodward
TRIPLETT, T. B. ..... Mooreland
VINCENT, DUKE ..... Vici
WALKER, HARDIN ..... Rosston
WINCHELL, F. Z. ..... Buffalo
WILLIAMS, C. E. ..... Woodward
WRIGHT, HERBERT ..... Supply